CLINICAL PROFILE ,ETIOLOGY,MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE CENTRE

Dissertation submitted to

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In partial fulfilment of the regulations for the award of degree of

M.D DEGREE (PEDIATRICS) BRANCH VII

INSTITUTE OF SOCIAL PEDIATRICS

STANLEY MEDICAL COLLEGE

CHENNAI – 600 001

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DECLARATION

I, **Dr.J.BALAJI** solemnly declare that the dissertation titled **"CLINICAL PROFILE,ETIOLOGY,MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE CENTRE"** was done by me at **Government Stanley Medical College during 2013- 2016** under the guidance and supervision of my chief **Prof. S.SHANTHI M.D, D.C.H.**

The dissertation is submitted to **The Tamilnadu Dr.M.G.R Medical University** towards the partial fulfilment of the rules and regulations for the **M.D. Degree Examination - BRANCH VII - in Pediatrics**.

Place : Chennai signature of the candidate

Date: **Dr. J.BALAJI**

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This is to certify that the dissertation titled **"CLINICAL PROFILE, ETIOLOGY, MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE CENTRE "** is a bonafide research work done under my guidance by **Dr.J.BALAJI** Postgraduate student,Department of Pediatrics, Government Stanley medical college, The Tamilnadu Dr.M.G.R Medical University, Chennai, in partial fulfilment of the requirement of the award for the degree of **M.D PEDIATRICS - BRANCH VII.**

Place : Chennai
Date :

Signature of the Guide
Dr.S.SHANTHI MD, DCH
PROFESSOR AND HOD
Institute of Social Pediatrics
Stanley Medical College,
Chennai - 600001

CERTIFICATE BY THE INSTITUTION

This is to certify that the dissertation titled **"CLINICAL PROFILE, ETIOLOGY, MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE CENTRE"** is submitted by **Dr.J.BALAJI** to **The Tamilnadu Dr.M.G.R Medical University, Chennai** in partial fulfilment of the requirement of the award for the degree of **M.D BRANCH VII (PEDIATRICS)** and is a bonafide work done by him under our direct supervision and guidance, during the academic year 2013 -2016

DR.SHANTHI MD, DCH
Professor and HOD
Institute of Social Pediatrics
Stanley Medical College
Chennai –600001

Prof .Dr.ISAAC CHRISTIAN MOSES MD
Dean
Stanley Medical College
Chennai –600001

Place: Chennai
Date:

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CLINICAL PROFILE,ETIOLOGY,MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE CENTRE

Dr J.Balaji postgraduate Pediatrics,Govt Stanley hospital

ABSTRACT :

Title : Clinical profile,etiology,management and outcome of serum electrolyte disturbances in children in the Pediatric Intensive Care unit in a tertiary care centre.

Aims and objectives: This prospective study evaluated the frequency, causes ,management and outcome pattern of sodium, potassium, calcium, chloride disturbances. Out of 227 patients studied from January 2015-august 2015,236 series (multiple episodes) of electrolyte disturbances were noted, most common electrolyte abnormality being hyponatremia (35.2% of cases) ,and is mostly of euvolemic type, the most common age group being 1month-1year,more in male children, commonest cause being seizures followed by poisoning. Not all children with electrolyte abnormality have symptoms or E CG abnormalities

Conclusion :Administration of routine maintenance fluids which are hypotonic may worsen hyponatremia.Electrolyte disturbances are common in PICU and high index of suspicion is needed,so that we can prevent electrolyte abnormalities and its complications.

Key words : electrolyte disturbances,sodium,potassium,calcium,chloride

CONTENTS

CLINICAL PROFILE,ETIOLOGY,MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PEDIATRIC INTENSIVE CARE UNIT IN A TERTIARY CARE CENTRE

INTRODUCTION :

Electrolytes are substances that ionizes when dissolved in suitable ionizing solvents

eg :water

Electrolyte abnormalities are common in children who need intensive care , they occur in variety of conditions, may remain unrecognized and result in morbidity and mortality irrespective of primary problem. Early recognition, a high index of suspicion and a thorough understanding of common electrolyte abnormalities is necessary to ensure their correction.

- Hyponatremia is particularly common in sick hospitalized children . It is invariably associated with hypo-osmolality and normal hydration and is attributed to SIADH. Acute hyponatremia poses an immediate danger to central nervous system.

- Hypernatremia occurs less frequently than hyponatremia on other hand, patients debilitated enough to develop hypernatremia carry a high mortality risk.

- A number of conditions predispose patients in the PICU to hyperkalemia such as renal insufficiency, adrenal insufficiency, insulin deficiency, resistance, tissue damage such as rhabdomyolysis, burns or trauma.

- The development of many electrolyte disturbances in PICU can be prevented by attention to use of intravenous fluids and nutrition.

- The development of acute renal failure continues to be a problem that markedly affects outcome in critically ill children.despite advances in treatment development of acute renal failure continues to be associated with high mortality rates .

- Delayed resuscitation will lead to ongoing kidney injury which in turn lead to established failure requiring various renal replacement therapies.

KNOWLEDGE GAP :

This study and the thesis presented here at Government Stanley Medical College Hospital PICU gains significance as there hasn’t been any of it’s kind prior to this.Hence survey regarding such electrolyte profile will lead to better understanding towards electrolyte disturbances in our Government Stanley Medical College PICU. Understanding such risks will enable us to provide appropriate fluid management and reduce the incidence of electrolyte disturbances in the PICU.

The osmolality and volume in the intravascular space are regulated by independent systems of water balance that in turn determines osmolality ,sodium homeostasis that determine the volume status.

AIMS AND OBJECTIVES

- To know most commonly occurring electrolyte abnormalities,etiology,management and outcome of serum electrolyte disturbances in sick children admitted to pediatric intensive care unit to Institute of Social Pediatrics Stanley medical college.
- To study the outcome pattern amongst the critically ill children seeking emergency care with electrolyte abnormalities with respect to underlying co-morbid conditions.

REVIEW OF LITERATURE:

- S.D. Subba rao. et al and Biju Thomas et al analysed 305 patients in St.john's hospital aged between 1 month and 14 years , who were admitted in PICU during the period. Ninety nine (32.45%) had electrolyte abnormalities. Of these 24 (7.9%) had mixed electrolyte imbalance.

- Hyperkalemia was the commonest found in 44 (14.4%)cases, hyponatremia was seen in 11 (3.6%) cases which is second commonest abnormality noted. Of the 99 patients with electrolyte imbalance, 24 (24.2%) expired. In these 24 patients 10 (41.6%) had hyperkalemia , 6(25%) had hyponatremia , 5(20.8%) had hypernatremia , 3(12.5%) had hypokalemia

- SV.S.S. Prasad , Sunit Singhi , K.S.Chugh et al analysed a total of 727 Children admitted in PICU the frequency distribution of serum sodium concentration in 727 children , hyponatremia was present in 217(29.8%) children while severe hyponatremia (serum sodium <125 mEq/l was found in 47(6.4%). Amongst those with severe hyponatremia in 21 the serum sodium ranged between 121-125 mEq/l. in 17 between 116-120 mEq/l and in 9 patients it was 115mEq/l or less. hypokalemia was found in 101 (13.9%) and 39 (5.4%) had hyperkalemia while In their study pneumonia and diarrheal disease, each accounted for about 20% cases of hyponatremia. The frequency was 26% in pneumonia and

33% in meningoencephalitic illness, being higher in summer as compared winter seasonal difference was observed

- 587 were normokalemic.

- Lamia. M Al Naama, Jawad Kadhum Abdul –Hassan et al, performed case control study on 150 children (87 boys and 63 girls), of age group between 2 months and 9yrs. 75 of them presented with acute CNS manifestations while the rest were considered as control.

- Eight of 75 pediatric patients (10.7%) with acute CNS diseases had hyponatremia syndrome, three were diagnosed with inappropriate antidiuretic hormone secretion. The highest percentage of hyponatremia (3 out of 6 patients) was found in patients with intracranial diseases. Four out of 38 patients(15.5%) presented with CNS infections

- Singhi S et al Indian pedia 1996 Jan 33(1.8%)9-14. 43(14) patients had 54 episodes of hypokalemia .predisposing factors included the nature of primary disease (renal disease 19%, acute diarrohea 14%,CCF & meningoencephalities 12% each). The over all mortality among patients with hypokalemia (25.6%) was significantly higher than that among the remaining PICU patients(10.9%), all the patients receiving rapid correction survived

- Cummings BM et al Intensive care medicine. 2013 june 6. A total of 512 patients had a potassium measurement. Of a total of 4484 potassium measurements, one third had abnormal values. Hypokalemia affected

40% of the admissions. Mild hypokalemia (3-3.4 mmol/l) affected 24% of the admissions. Moderate or severe hypokalemia (k<3.0mmol/l) affected 16% of admissions.

- Hyperkalemia affected 29% 0f admissions. Mild hyperkalemia (5.1 – 6.0 mmol/l) affected 17% of admissions. Moderate or severe hyperkalemia (>6.0 mmol/l) affected 12%. On univariate analysis, severity of hypokalemia was associated with mortality

- Hoorn et al found a 30% overall incidence of pNa less than 136mmol/l and a 38% incidence in the ICU. Severe hyponatriemia PNa less than 125mmol/l, was present in 3% of hospitalized patients and occurred during hospitalization in fully half of cases of the cases

About two thirds of the human body is made up of water. In an infant and child total body water is 65%.As the child grows older, TBW decreases to 60% in male and 55% in female. The percentage of intracellular fluid compartment remains the same in all age groups as 40%.Fall in TBW is reflected in ECF.

Sodium disturbances :

Normal sodium levels 135-145 meq/L

Serum sodium <135meq/L – hyponatremia

Serum sodium >135meq/L- hypernatremia

- Sodium Bulk cation of extracellular fluid → change in S_{Na} reflects change in total body Na^+
- Principle active solute for the maintenance of intravascular & interstitial volume
- Absorption: throughout the GI system via active Na,K-ATPase system

  Excretion: urine, sweat & feces
- Kidneys are the principal regulator
  - 2/3 of filtered Na^+ is reabsorbed by the proximal convoluted tubule, increase with contraction of extracellular fluid
  - Countercurrent system at the *Loop of Henle* is responsible for Na^+ (descending) & water (ascending) balance – active transport with Cl^-
  - Aldosterone stimulates further Na^+ re-absorption at the distal convoluted tubules & the collecting ducts
  - <1% of filtered Na^+ is normally excreted but can vary up to 10% if necessary
- Major component of serum osmolality
  - $S_{osm} = (2 \times Na^+) + (BUN / 2.8) + (Glu / 18)$
  - Normal: 285-295
- Alterations in S_{Na} reflect an abnormal water regulation.

Hyponatremia :

The age and sex of the child does not affect the frequency of hyponatremia but a significant seasonal difference was apparent in the present study. Incidence of hyponatremia was higher in summer as compared to winter existed through most of the diagnostic categories,one exception being diarrhea.Higher amount of water and salt loss through sweating might have contributed to this differences. The observation implies suspicion of hyponatremia in summer could be enormously increased and this should be anticipated .The commonest cause of low sodiumdue to bronchopneumonia,diarrhea,meningitis with encephalitis.

In contrast to this, the frequency that was reported as hyponatremic dehydration was only 10% in children with acute diarrhea in the western countries.this lead to prediction that hyponatremia in diarrhea may be hypovolemic type that may be due to excessive sodium loss in gastro-intestinal secretions; intake of salt free drinks and increased loss of salt through sweating in our climate might have contributed.

Apparently, hyponatremia occurs frequently without having significant changes in extracellular fluid volume in children with infectious diseases needing hospitalisation and needs to be looked into and should be managed appropriately.

Symptoms of hyponatremia include nausea, abdominal cramps, vomiting, headache Edema ,muscle weakness, tremor, paralysis, disorientation, slowed breathing, seizures coma.

Studies on the mechanism of hypotonic hyponatremia with euvolemia have showed a role of syndrome of inappropriate ADH secretion (SIADH) in those children. Almost every patients having hypotonic hyponatremia with euvolemia could be classified as having SIADH as these patients had normal hydration status, decreased plasma osmolality , increased urine osmolality and normal renal function.There is inappropriately higher concentration of plasma vasopressin than that is expected for the degree of hyposmolality has been demonstrated in asso-ciation with euvolemic hyponatremia in children with meningitis, asthmaand hospitalized adults.

Thus, ADH mediated renal salt loss and water retention could be the cause of hyponatremia in such patients. However, severehyponatremia may occur in association with SIADH without unusual loss of sodium from the body or dilution of the plasma sodium. Evidence supporting redistribution and accumulation of sodium within the cells has also been presented. Recently, Hannon and Bos-ton have shown significant intracellular shifts of sodium chloride and water in septic animals. They suggested that in these animals hyponatremia and hypoosmolality was caused by a combination of intracellular shift of sodium and water, and dilution of extracellular space probably as a result of physiological antidiuretic hormone secretion.

Data from our ongoing research suggest a significant increase in RBC sodium in the presence of hyponatremia in septicemic children. Further studies are needed to clarify the mechanism responsible for hyponatremia in acute infections. In conclusion, hyponatremia occurs frequently and should be looked for in all sick children. It is of hypotonic-euvolemic type in almost all the acute infections except diarrhea and should be managed accordingly.

HYPERNATREMIA

Hypernatremia defined as serum sodium >145 meq/l have a deficit of water in relation to the body sodium stores, net water loss or a hypertonic sodium gain. most cases are due to net water loss, where as hypertonic sodium gain results from sodium loading accidentally or some interventions.Increased sodium in extracellular compartment leads to increased plasma tonicity, this in turn leads to water movement across cell membrane result in cellular dehydration.

Mechanism leading to hypernatremia or along with the same includes the following

- Water depletion eg:diabetes insipidus
- Depletion of water exceeds depletion of sodium eg:diarrhea
- Excess of sodium eg:salt poisoning

the risk is highest among infants and ventilated patients.

Infants with large surface area with respect to height or weight in comparison to adults wil have a large evaporative water loss. Infants with

diarrhea and infant formula that is improperly prepared or inadequate infant mother interaction during breastfeeding have higher incidence of hypernatremia. There will be increased efflux of water from cellular compartment inorder to maintain osmolality that is equal inside and outside the cell.cell contraction make the brain cells excessiblyvulnerable.Therefore hypernatremic dehydration causes shrinkage of brain, tear cerebral bloodvessels, cerebral hemorrhage, paralysis, seizures and encephalopathy.

Rapid rehydration of hypernatremic patients with hypotonic fluids may cause cerebral edema that can lead in coma, seizures or death.

Causes of hypernatremia include ,

1) hypovolemic hypernatremia due to increased loss of hypotonic fluids

Increased loss of hypotonic fluid coupled with insufficient water intake is the commonest cause of hypernatremia in children.

There is decreased total body sodium ,body content of water is decreased further.

There is shift of water from ICF to ECF as a compensation to increased serum osmolality. Here the circulating volume is maintained til there is marked water deficits. Osmotic diuretics like glycerol or mannitol and diuresis in diabetes mellitus can cause hypernatremia due to increased urine losses.children with hypernatremic dehydration due to nonrenal losses have urine that is concentrated with low urine sodium content.

2) Hypernatremia due to water deficit :

Central or nephrogenic diabetes insipidus, excessive sweating, fever, sustained hyperventilation, urine is concentrated with low urine sodium.

3) Hypernatremia due to excess sodium euvolemic hypernatremia :

Seen in solutions for rehydration with increased sodium,concentrated formula,iatrogenical,urine concentrated normally,with increased sodium losses.

Potassium disturbances :

Potassium values normal range 3.5-4.5 meq/L

- Largely contained intra-cellular → S_K does not reflect total body K
- Important roles: contractility of muscle cells, electrical responsiveness
- Principal regulator: kidneys
- Complete absorption in the upper GI tract
- Kidneys regulate balance
    - 10-15% filtered is excreted
- Aldosterone: increase K^+& decrease Na^+ excretion
- Mineralocorticoid & glucocorticoid → increase K^+& decrease Na^+ excretion in stool
- Solvent drag
    - Increase in S_{osmo}→ water moves out of cells → K^+ follows

- 0.6 S_K / 10 of S_{osmo}
- Evidence of solvent drag in diabetic ketoacidosis

- Acidosis
  - Low pH → shifts K^+ **out** of cells (into serum)
  - Hi pH → shifts K^+**into** cells
  - 0.3-1.3 mEq/L K^+ change / 0.1 unit change in pH in the opposite direction

Causes of hyperkalemia

Spurious

Difficult blood draw → hemolysis → false reading

Increase intake

Iatrogenic: IV or oral

- Blood transfusionsDecrease excretion
  - » Renal failure
  - » Adrenal insufficiency or CAH
  - » Hypoaldosteronism
  - » Urinary tract obstruction
  - » Renal tubular disease

» ACE inhibitors

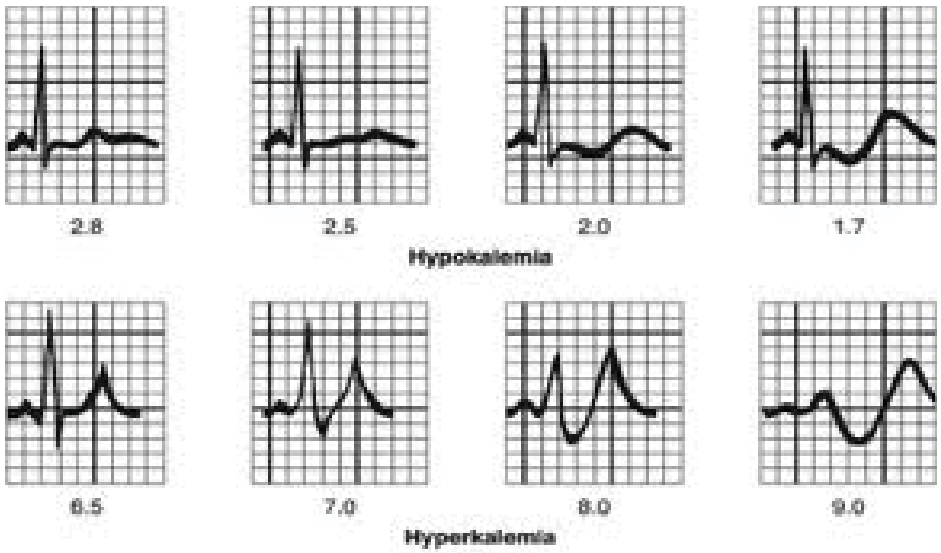
» Potassium sparing diureticsTrans-cellular shifts

» Acidemia

» Rhadomyolysis; Tumor lysis syndrome; Tissue necrosis

» Succinylcholine

» Malignant hyperthermia



HYPERKALEMIA :

– Intestinal ischemia shock was induced either by temporary occlusion of the three splanchnic arteries for 40 min (SAO-shock) or by temporary occlusion of the portal vein for 35-40 min (PVO-shock). In both types of shock, life can be considerably prolonged (5-8-fold) by treatment with rat plasma plus glucose. Eventually, death is caused by heart failure due to hyperkalemia (plasma K+ concentration greater than 10 mmol/l). The

amount of K+ causing this hyperkalemia is estimated at roughly 10% of the total body K+. Acidosis, low blood pressure, reduced kidney function, and disintegration of erythrocytes in the gastrointestinal (GI) tract probably are of no or the skeletal muscles, or the erythrocytes release K+. Although the K+ concentration of the contents of the GI tract as well as the K+ transport by the portal vein were increased, the source of the excess K+ remains obscure. Removal of the contents of the stomach and small intestine, followed by flushing of the gastrointestinal tract, may have a favorable effect on the course of plasma K+ (and plasma glucose) concentration, indicating that toxic products from the damaged intestines may be important lethal factors

- minor importance in causing this extreme hyperkalemia. No indication was found that the liver, the skeletal muscles, or the erythrocytes release K+. Although the K+ concentration of the contents of the GI tract as well as the K+ transport by the portal vein were increased, the source of the excess K+ remains obscure. Removal gastrointestinal tract, may have a favorable effect on the course of plasma K+ (and plasma glucose) concentration, indicating that toxic products from the damaged intestines may be important lethal factors.
- Clinical features of hyperkalemia includes weakness,nausea,abdominal pain,irregular heart beat,diarrhea,muscle pain.

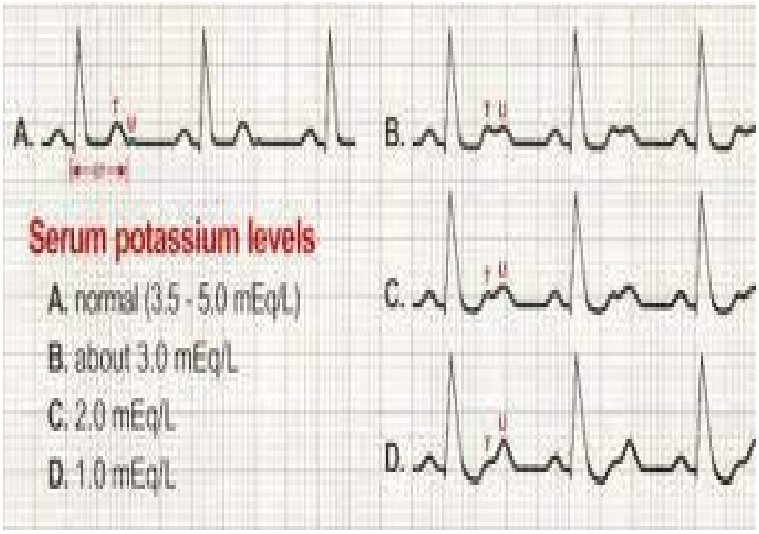
HYPOKALEMIA :

POTASSIUM disturbance especially hypokalemia, during admission was known to occur in a number of patients . Low potassium level can have effects that are profound on electrical activity in cardiac, skeletal and smooth muscle. These disturbances if severe, result in life threatening conditions like arrhythmias, cardiac-arrest, respiratory failure, paralytic ileus and paralysis of muscles.Hypokalemia appears to be one of the most common electrolyte disturbances in sick children. Studies addressing its incidence and outcome are few and chiefly from developed countries.

Hypokalemia is common in critically ill children and have a significantly higher mortality. Patients having preexisting kidney disease, septicemia,bronchial asthma, cardiac disease with congestive cardiac failure, diarrhea that is severe and meningoencephalitis were most likely to show evidence of hypokalemia. The apparent cause for occurance of hypokalemia in most patients being a loss of potassium from the body through gastrointestinal or urinary tract , because of underlying disease process, or because of associated use of drugs like diuretics,corticosteroids and asthma drugs. In a patient with DKA hypokalemia would be due to correction of acidosis and insulin use . In critically sick children hypokalemia may be caused by endogenous massive release of epinephrine.

Hypokalemia Causes

- Distribution from ECF
  - » Hypokalemic periodic paralysis
  - » Insulin, B-agonists, catecholamines, xanthine
- Decrease intake
- Extra-renal losses
  - » Diarrhea
  - » Laxative abuse
  - » Perspiration
- Excessive colas consumption
- Renal losses
- DKA
- Diuretics: thiazide, loop diuretics
- Drugs: amphotericin B, Cisplastin
- Hypomagnesemia
- Alkalosis
- Hyperaldosteronism
- Licorice ingestion
- Gitelman & Bartter syndrome



Hypokalemia

- Flattened or inverted T-wave
- U wave: prolonged repolarization of the Purkinje fibers
- Depressed ST segment and widen PR interval
- Ventricular fibrillation can happen

Symptoms of hypokalemia include weakness,paralysis,increased urination,arrhythmias,orthostatic hypotension,muscle pain ,tetany

Calcium disturbances :

Normal range of total calcium is 8.8-10.8 mg/dl (2.2-2.7mmol/L)

Normal range of ionized calcium is 4.2-5.5mg/dl(1-1.4mmol/L)

HYPOCALCEMIA :

Total calcium <8mg/dl or ionized calcium <1mmol/L seen in seriously ill children.ionized calcium if possible should be measured.

Causes for hypocalcemia :

Multiple transfusion –chelation of calcium by citrate.ionized calcium is low

But total calcium is normal.high alkalosis causes more calcium to be bound to albumin leading to decreased ionized calcium.increased phosphate,septic shock,

Rhabdomyolysis,tumourlysis,acutepancreatitis,hypoparathyroidism,

Pseudohypoparathyroidism,decreased magnesium levels,nutritional like malabsorption,hepatic diseases,antiepileptics therapy,kidney failure.

Clinical features of hypocalcemia :neuromuscular activity like seizures,tremors,laryngospasm,carpopedal spasm,muscle cramps.

Hypotension,cardiac failure.increased risk of digitalis toxicity,sapsi like syndrome.

Calcium is the most abundant mineral in the body. 99% of the total body calcium is stored in bone, and serum levels constitute less than 1%. Various factors regulate the homeostasis of calcium and maintain serum calcium within a narrow range. These include parathormone (PTH), vitamin D, hepatic and

renal function (for conversion of vitamin D to active metabolites), and serum phosphate and magnesium levels.

Serum calcium is present in two forms: the free (ionized) and the bound form. Only about 50% of circulating calcium is present in the physiologically free form. The rest is either bound to proteins (40%) or complexed (10%) with bicarbonate, citrate, and phosphate. The ionized calcium level varies based on the level of serum albumin, blood pH, serum phosphate, magnesium, and bicarbonate levels, the administration of transfused blood containing citrate and free fatty acid content in total parenteral nutrition. The normal range for ionized calcium is 1-1.25 mmol/L (4-5 mg/dL).

The concentration of calcium in the serum is critical to many important biologic functions, including the following:

- Calcium messenger system by which extracellular messengers regulate cell function
- Activation of several cellular enzyme cascades
- Smooth muscle and myocardial contraction
- Nerve impulse conduction
- Secretory activity of exocrine glands

Symptoms of hypocalcemia :

Hypocalcemia manifests as central nervous system (CNS) irritability and poor muscular contractility. Low calcium levels decrease the threshold of excitation of neurons, causing them to have repetitive responses to a single stimulus.

Because neuronal excitability occurs in sensory and motor nerves, hypocalcemia produces a wide range of peripheral and CNS effects, including paresthesias, tetany (i.e., contraction of hands, arms, feet, larynx, bronchioles), seizures, and even psychiatric changes in children.

Muscle excitability is depressed because hypocalcemia impedes acetylcholine release at neuromuscular junctions and, therefore, inhibits muscle contraction. However, the increase in neuronal excitability overrides the inhibition of muscle contraction. Cardiac function may also be impaired because of poor muscle contractility.

Hypercalcemia :

Total calcium >10.5mg/dl or ionized calcium >1.2mmol/L

Hypercalcemia seen in oncology patients, primary hyperparathyroidism prolonged immobilization.

Symptoms of hypercalcemia :

CNS- slowed nerve conduction :CNS depression,fatigue, seizures and coma

CVS- arrhythmias ,ventricular ectopy,myocardial depression

GIT – constipation,nausea,vomiting,peptic ulcer disease

RENAL – impaired renal tubular defects,concentrating defects

- ***Hypercalcemia:*** Clinical presentation
  - Groans: constipation
  - Moans: psychic moans (fatigue, lethargy, depression)
  - Bones: bone pain
  - Stones: kidney stones
  - Psychiatric overtones: depression & confusion
  - Fatigue, anorexia, nausea, vomiting, pancreatitis
  - ECG: short QT interval, widened T wave

Chloride :

- with Na+ and H2O to help maintain cellular integrity, fluid balance and osmotic pressure
- Affects acid/base balance (enzyme activator, serves as buffer in exchange of O2 and CO2 in RBC's)
- Major ECF anion
- Serum level: 95-108 mEq/L

Function; circulates In conjunction with Ca+, Mg+, helps maintain nerve transmission/muscle function

- Vital role in production of HCL

- Obtained primarily from foods (processed) and table salt, daily need: ~750mg.90% excreted by kidney

MAINTENANCE FLUID AND ELECTROLYTES

-Based on the caloric expenditure model, each calorie expended requires provision of water in the ratio of 1 ml/cal metabolized/day at rest.

-Also, according to the caloric expenditure model maintenance sodium and potassium ranges/100 ml of maintenance fluid/day have been determined. Upper limit has been chosen for sodium and lower limit has been chosen for potassium to be placed in the “Segar box”.

-Common names for commercially available saline solutions and their sodium concentrations are:

Normal saline (0.9% NaCl/L) 154 mEq Na+/L

One-half normal saline (0.45% NaCl/L) 77 mEq Na+/L

One-third normal saline (0.33% NaCl/L) 57 mEq Na+/L

One-quarter normal saline (0.2% NaCl/L) 34 mEq Na+/L

Ringer’s lactate 130 mEq Na+/L

(Contains 4 mEq K+, 109 mEq Cl-, 28 mEq bicarb equivalent all/Liter, and 3mg/dl of Ca++)

-Addition of glucose to each of the above at a minimum of 5 gm/100 ml (5% dextrose) minimizes tissue catabolism to the point that protein stores

are somewhat “spared” from providing substrate for gluconeogenesis. Ketosis from fat metabolism is also prevented.

An intravenous fluid is the commonest intervention administered to a

Hospitalized children. The indications for administering intravenous fluids

are to either expand a contracted extracellular fluid or as maintenance fluids to

replace urine output and insensible water loss in a fasting patients.the administration of IVF is considered as an invasive procedure and should be dealt with the same respect and vigilance as any drug prescription.

Objective of intravenous fluid therapy is to fill the intravascular compartment. Fluids that are isotonic include normal saline NS or Ringers lactate RL will be retained in the vascular compartment more than other fluids.

Fluids that are hypotonic include ½ GNS, ¼ or 1/5GNS whwn administered will stay in vascular compartment in small quantities. Therefore isotonic fluids are preferred in correction of shock and deficit.

Source of water loss	Estimated water loss in ml/100Kcal/day
Insensible	
Skin	30
respiratory	15
Sensible	
Stool	10
Minimal sweating	10
urine	50

Table no.1,source of water loss and amount of water loss

Osmolality :

Osmolality is the number of osmotically active particles present in a solution per kilogram of solvent.

Osmolarity :

Osmolarity is the number of osmotically active particles present per litre of solution.

These two terms are often used interchangeably.

Tonicity :

Tonicity is the effective osmolality of a solution and is equal to the sum of the concentrations of solutes that have capacity to exert an osmotic force across a

semipermeable membrane that is impermeable solutes eg sodium.Tonicity is the property of the solution with reference to its membrane.osmolality and osmolarity both is the property of a solution independent of any membrane because it includes both impermeable and permeable solutes like urea.

One example 5%dextrose is isoosmolar initially with plasma but in normal conditions,dextrose is permeable across the membrane and an ineffective solute that readily enters the cell. Therefore 5% dextrose is isoosmolar with plasma but hypotonic with reference to cell membrane.

The most potent stimuli for ADH secretion are an increase in serum osmolality, hypovolemia and hypotension. Non osmotic stimuli like pain, drugs, anesthetic agents, stress, nausea, and vomiting can also cause increase in ADH secretion .ADH checks the renal water excretion even if there is low plasma osmolality. This impaired ability to excrete hypotonic urine coupled with positive balance of electrolyte free water leading to hyponatremia. Therefore administration of hypotonic fluids will exacerbate the fall in sodium concentration.

Sodium is the main cation in the extracellular fluid,thereby determining the ECF volume.An isotonic solution will have approximately 154 meq/L monovalent cations .Normal tonicity is 270-290 mosm/kg.Drastic variation in the tonicity of the extracellular compartment causes water shifts that leads to neurological consequences.

Sodium determines the extracellular fluid volume ,regulates the movement of water across the cell membrane thereby determining intracellular edema that occurs in the presence of hyponatremia.Children have a larger intracellular fluid volume per total skull volume,therefore greater risk of neurological sequelae secondary to hyponatremia.No single fluid rate or composition is ideal for all children ,however isotonic solutions may be the most physiological and therefore the safest empirical choice for maintenance IV fluids in the critical care setting.

The choice of a solution in the acute setting should not be for the purpose of satisfying the calculation of the daily sodium or caloric requirements for a healthy child ,but should aim to maintain tonicity balance in the acute phase of illness and in the postoperative period when the patients are at highest risk of fluid and electrolyte abnormalities, in particular hyponatremia.Excretion of electrolyte free water is limited in these patients and thus further administration of exogenous electrolyte free water in the form of hypotonic solutions increases the risk of acute hyponatremia and its associated morbidity.

Isotonic intravenous fluids should be considered or hypotonic solutions should be avoided /contradicted in patients for whom a higher effective osmolality needs to be maintained or a fall in effective osmolality should be avoided during their critical period of illness , eg .CNS injury ,Diabetic keto acidosis. Isotonic solutions are also indicated in the postoperative period and in patients with gastroenteritis,especially when accompanied by evidence of elevated urinary tonicity. Hypotonic solutions should be given if there is a need to create

a positive balance for electrolyte free water when there is an occasion of electrolyte free water deficit that may occur with large water or osmotic diuresis,or if there is non renal loss via the gastrointestinal tract or skin.

Fluids used for hydration contain higher concentrations of chloride that induce or increase hyperchloremia and metabolic acidosis and decrease glomerular filtration rate and kidney vasoconstriction .

Stewarts theory states that 3 independent variables determine the pH , in manner of changing the degree of dissociation of water into hydrogen ions . 3 variables include strong ion differences,pCO2,the charge from weak acids.

A decrease in strong ion differences or an increase in the Pco2,or the charge from weak acids has an acidifying effect on plasma.one of the effect that plasma chloride has on Ph, can be assessed after looking the strong ion differences that is calculated as the charge differences between the sum of measured cations and measured anions .A strong ion is defined as the one that is almost completely dissociated at physiological Ph,.As both Na+ and cl- are the major strong ions in plasma their relative ratio to one another largely determines the strong ion differences.

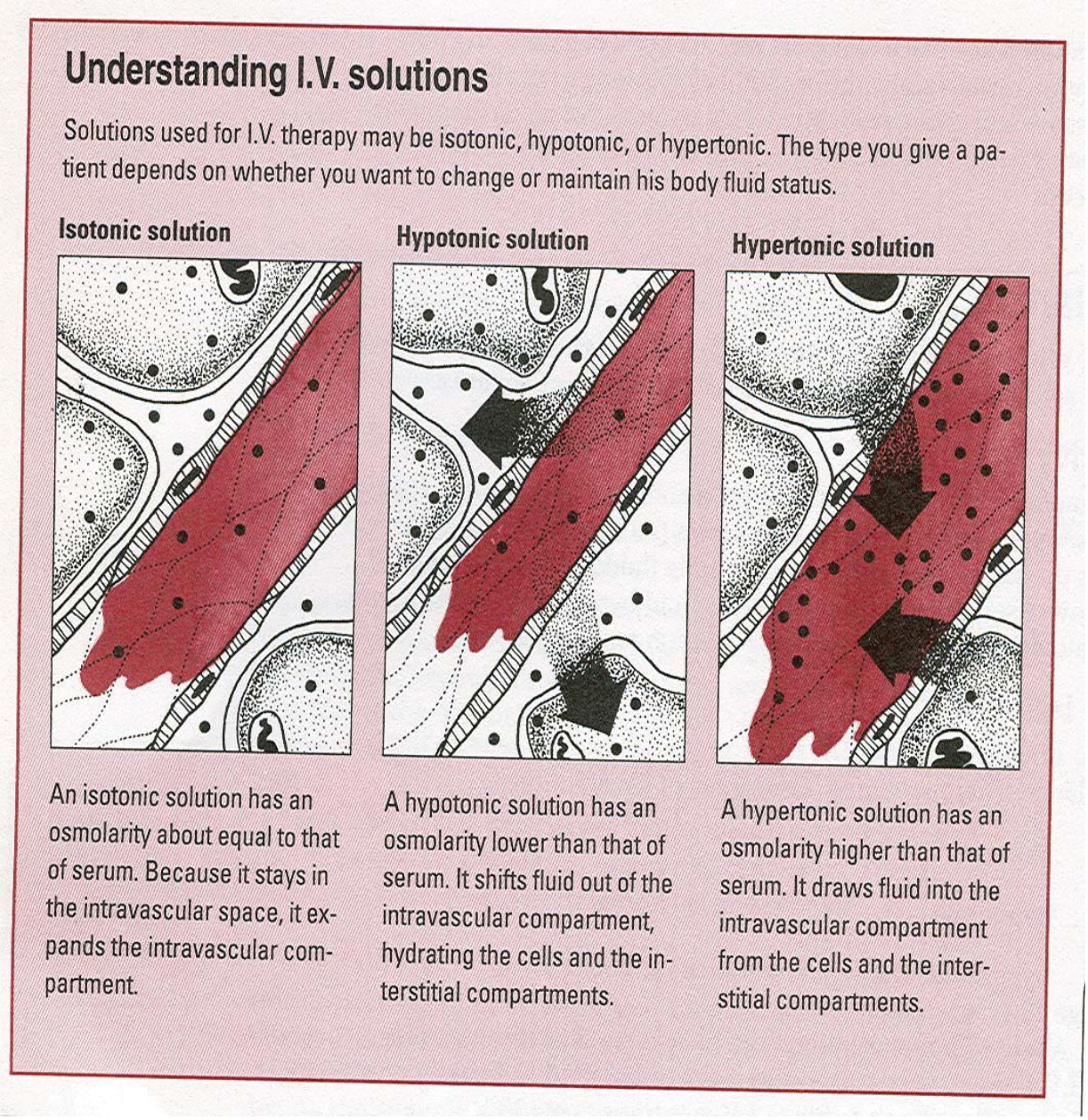


Figure no.1,type of fluid and its relation to vascular compartment

An increase in the plasma chloride to sodium will reduce the plasma strong ion differences ,hence increasing the dissociation of water into hydrogen ions.That is the smaller the strong ion differences ,lower the pH .Therefore 0.9% Normal saline is in equimolar distribution of sodium and chloride and therefore has an strong ion difference of 0.The administration of large quantities of normal

saline will reduce the strong ion differences thereby producing a hyperchloremic metabolic acidosis.

Both normal saline and colloid preparations such as 5% albumin have an acidifying effect on plasma and therefore are not physiological.Ringer lactate,Hartmann's and plasmalyte solutions with multicarbon anions that contain choride and sodium in the concentration similar to plasma are more physiological and may be less likely to acidify the plasma.

Ideal maintenance fluid should be Balance solutions that are isotonic to plasma and strong ion diffences must not be altered. To tackle the problem of atmospheric CO2 loss,most manufacturers of balanced salt solutions substitute organic anions for HCO3.Example include L-Lactate,acetate,gluconate malate and citrate.There are particular considerations unique to critically sick child that need a reduction as much as 40-50% of the currently recommended volumes,once the patient is intravascular replete.

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METHODOLOGY

- **Duration of study**: January2015 to August2015
- **Sample size and design**: Two hundred and twenty seven Children admitted in PICU over a period of 8 months fulfilling inclusion and exclusion criteria.
- **Study design** : cross sectional study
- **Study place**:

  The Pediatric Intensive Care Unit
  Institute of Social Pediatrics
  Government Stanley Medical College
  Chennai – 600 001

- **Study population** :

Children of the age group between 1 month and 12 years admitted in the PICU.

Inclusion criteria :

- >1 month to 12 years age , both sexes
- All children admitted in PICU

Exclusion criteria :

- Those who have not given consent.
- Post cardiac arrest resuscitated child

Sample size :

Cross sectional study for prevalence

Sample size is calculated using CDC Atlanta widely used by statistician

Sample size calculation :

Sample Size for Frequency in a Population

Population size(for finite population correction factor or fpc)(*N*):	500
Hypothesized % frequency of outcome factor in the population (*p*):	50%+/-5
Confidence limits as % of 100(absolute +/- %)(*d*):	5%
Design effect (for cluster surveys-*DEFF*):	1

Sample Size(*n*) for Various Confidence Levels

ConfidenceLevel(%)	Sample Size
95%	218
80%	124
90%	176
97%	243
99%	286
99.9%	343
99.99%	377

Equation

Sample size $n = [DEFF*Np(1-p)]/ [(d^2/Z^2_{1-\alpha/2}*(N-1)+p*(1-p)]$

Results from OpenEpi, Version 3, open source calculator--SSPropor

Sample size taking 10% as nonresponse=0.9%

Previous study shows prevalence of hyponatremia to be 200

200/0.9 =222.2 sample size =222 (225)

Based on the previous study ,the sample size of the present study is calculated to be 222

METHOD OF STUDY

This study of electrolyte disturbances was conducted in the PICU of a tertiary care referral hospital attached to a medical college in NorthChennai. This was done for a period of 8months. This hospital caters to the medical needs of a population, the majority of whom belong to a lower socio-economic status. Being a referral institute with numerous feeding hospitals in the surrounding locality, the patient turnover in the PICU is very rapid. Hence it is only the very sick children who get admitted to thePICU.

Ethical clearance for the study was obtained from the Institutional ethics Committee.

Informed written consent was obtained from the parent of the child included in the study. Detailed history and clinical examination of all the patients taken up for the study were done at the time of admission to the PICU as shown in the Proforma. Routine laboratory investigations done .

Electrolyte analyser machine

Figure no.2,electrolyte analyser machine

Time of collection:

At the time of admission the patients clinical picture is recorded in prefixed proforma. Venous blood sampling is obtained from each patient enrolled in the study and is sent for estimation of Electrolytes, Blood urea, Glucose levels. Serum osmolality (calculated), Urine osmolality, Urine spot sodium,potassium,were done in selected patients. Imaging studies as relevant to the admission diagnosis

Measurement of electrolytes

Electrolytes are measured by a process known as potentiometry. This method measures the voltage that develops between the inner and outer surfaces of an ion selective electrode. The electrode (membrane) is made of a material that is selectively permeable to the ion being measured. For example, sodium electrodes are made from a special glass formula that selectively binds sodium ions.

- The inside of the electrode is filled with a fluid containing sodium ions, and the outside of the glass membrane is immersed in the sample.
- A potential difference develops across the glass membrane that is dependent upon the difference in sodium concentration (activity) on the inside and outside of the glass membrane.
- This potential is measured by comparing it to the potential of a reference electrode. Since the potential of the reference electrode is held constant, the difference in voltage between the two electrodes is attributed to the concentration of sodium in the sample. Ion selective membranes can be made from materials other than glass.

OBSERVATION AND RESULTS

Analysis of the data collected has been done and statistical significance of the risk factors are established as discussed in the following section.

During the study period from January 2015 to august 2015, the total number of PICU admission was 227 patients. Among 227 patients ,236 episodes of serum electrolyte disturbances were noted.

SODIUM DISTURBANCES :

Among this 227 children studied, 85 children had sodium disturbances that included both hyponatremia(80 children) and hypernatremia(5 children) depicted in the pie chart. This number accounts for 37.44% of the patients admitted to PICU.

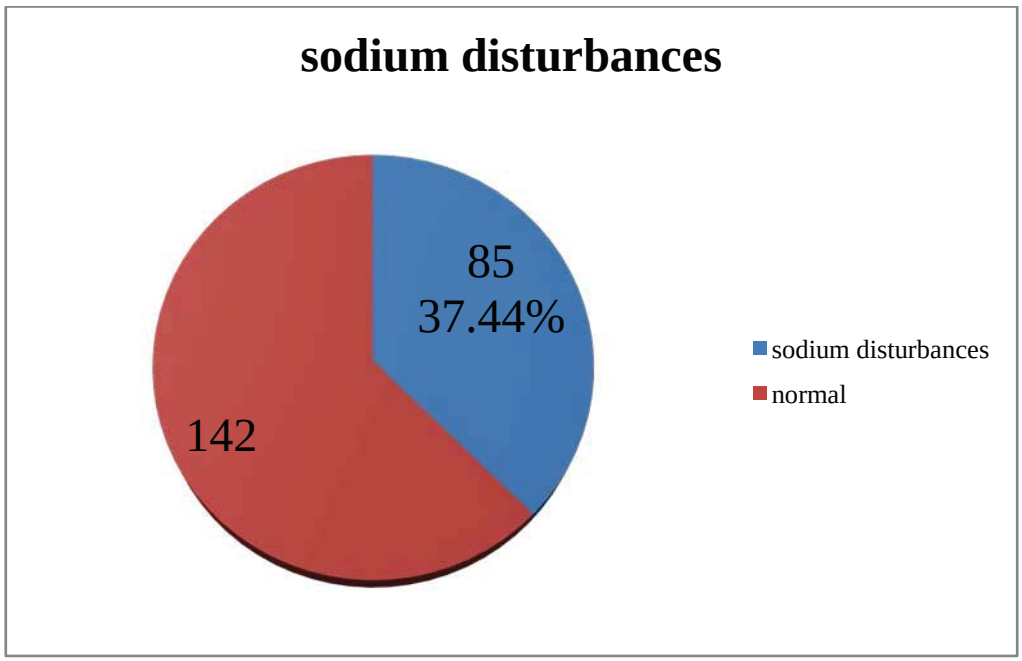


Figure no.3,number of sodium disturbances and normal electrolyte values

Age distribution :

Of the children with sodium disturbances, distribution based on age were analysed and the corresponding chart is shown as below.Maximum number (27 children) of patients who developed sodium disturbances were in the age group of 1month to 1year and the mean age in years of the study population was calculated to be 1month -2yrs,most common electrolyte imbalance being hyponatremia 80 cases.

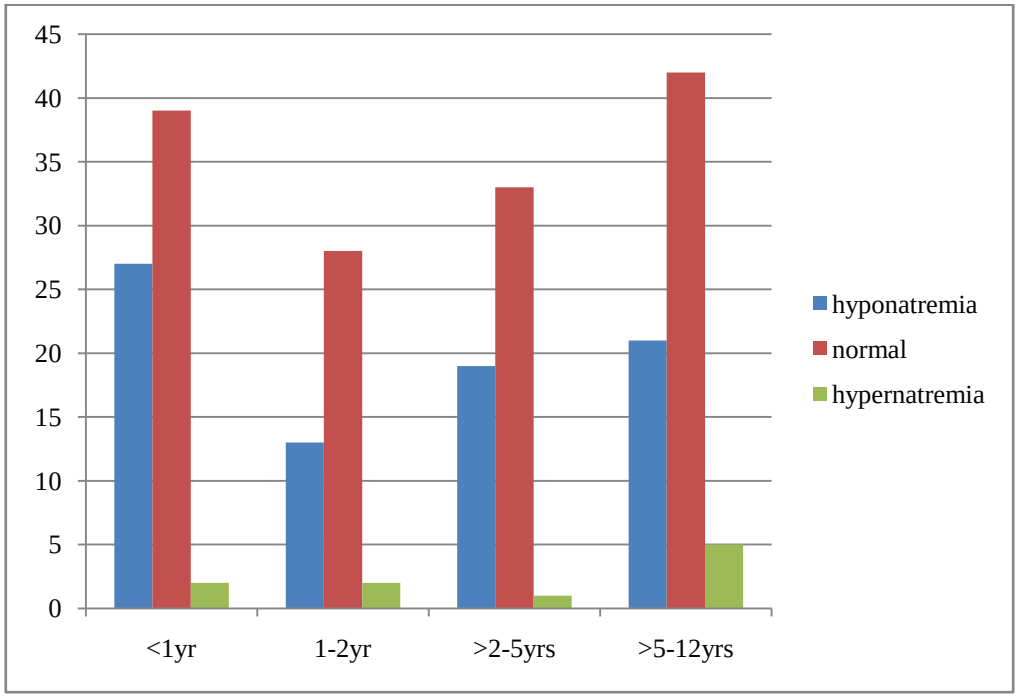


Figure no.4,Incidence of sodium disturbances age wise distribution

Among 80 cases of hyponatremia, Incidence of hyponatremia is more common among infants(about 27 cases).Among 5 cases of hypernatremia , it is more common among children aged >5 yrs.

Incidence of hyponatremia is least among 1-2yrs.on the contrary incidence of hypernatremia is least among age group of 2-5yrs

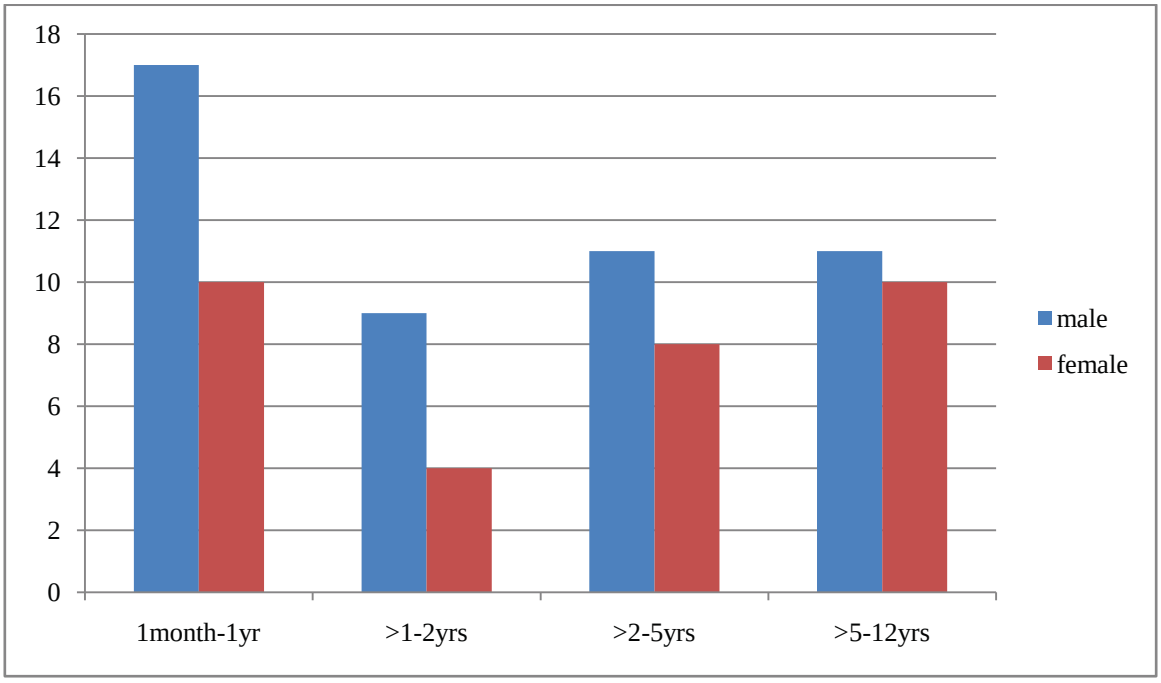


Figure no.5,**Sex wise distribution of hyponatremia**

Total number of hyponatremia was 80 children

Incidence of hyponatremia is more common among male child about 48 cases,most common age group being 1month-1year (17cases). Out of the 80 patients who developed hyponatremia 48 patients were male that is 60 % and 32 patients were females that is 40 % were female with male :female ratio 1.5:1.

ETIOLOGY AND SODIUM DISTURBANCES

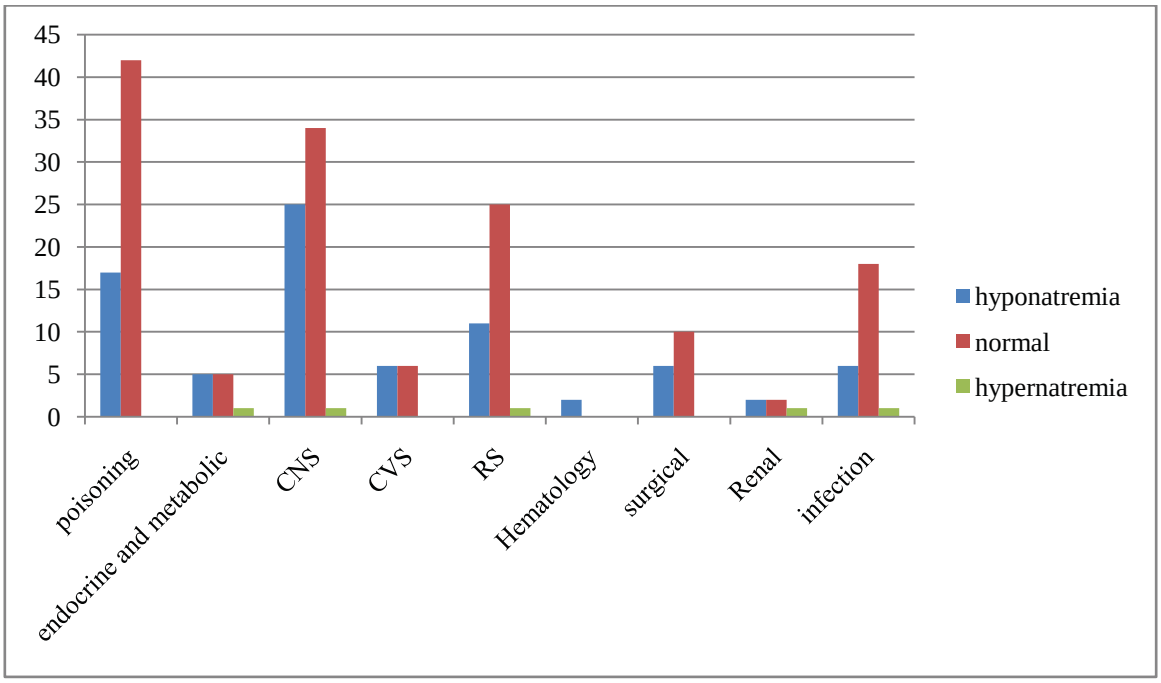


Figure no.6: Etiology and Sodium abnormalities

The most common cause of hyponatremia is CNS disorder 25 patients followed by poisoning 17 patients forming 31.25% and 21.25 % respectively.

Poisoning and CNS disorders accounted for 21.25% and 31.25% of hyponatremia respectively; others were accounted for by bronchopneumonia (13.75%) septicemia (7.5%), and renal(2.5%),cardiac(7.5%).

Poisoning includes kerosene poisoning,liquid detergent poison,camphor insecticide poisoning,

Endocrine /metabolic causes include diabetic ketoacidosis,metabolic liver disease,hepatic encephalopathy,neuronal ceroidal lipofuschinosis

CNS causes include seizure disorder,acute CNS infection

CVS causes include cyanotic/acyanotic heart diseases,cardiomyopathy,arrhythmias

RS causes include bronchopneumonia/bronchiolitis,aspiration pneumonia ,acute severe asthma

Hematological causes include sickle cell anemia .malignancies,hemolytic anemia

Surgical causes include post VP shunt ,head injury,hepatoma,extrahepatic biliary atresia,thoracotomy/ICD pneumothorax

Endocrine ,CNS disorders,Respiratory system,renal, infection each contributing 1 case for hypernatremia.the

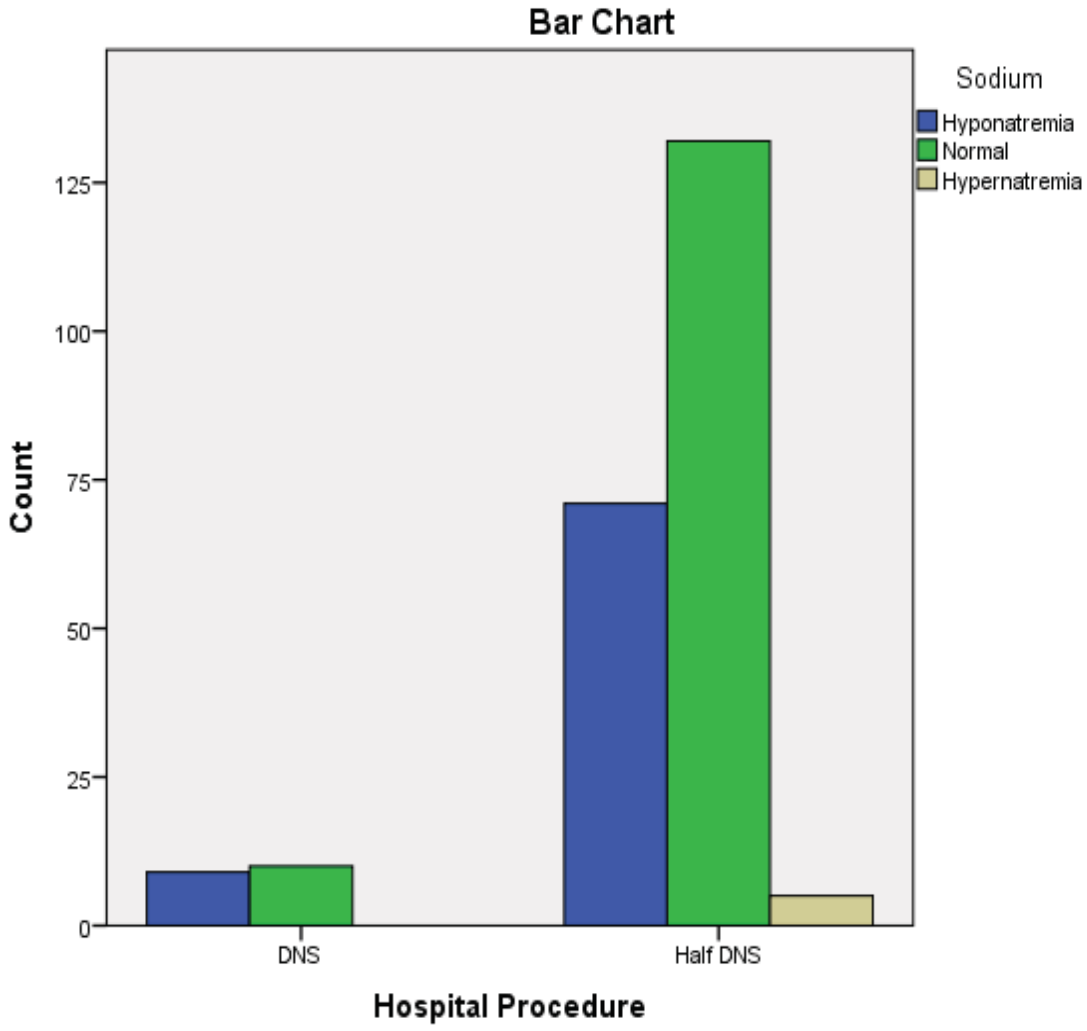


Figure no.7,maintenance fluid and sodium abnormalities

9 patients with hyponatremia received DNS that is about 11.25% of

hyponatremia,71 patients received ½ DNS 88.75% had hyponatremia.

Most common age group of hyponatremia was 1month-1yr (33.75%)

Clinical evaluation and concurrent plasma and urinary osmolality and urine sodium suggested that hyponatremia associated with pneumonia, meningitis/encephalitis, septicemia, seizures and miscellaneous diseases was of euvolemic (dilutional) type in more than 80% patients while in all children with acute diarrhea it was of hypovolemic type. The study has shown that hyponatremia occurs frequently in sick children requiring emergency care, and should receive appropriate attention in the management plan.

The patients were grouped on the basis of serum sodium concentration into normonatremic (serum sodium > 135mEq/L) and hyponatremic (serum sodium <135mEq/L or less). Serum sodium concentration of 125 mEq/L or less was classified as severe hyponatremia. Hyponatremia was further categorized into five types: euvolemic (normal hydration, plasma osmolality ≤280 mOsm/kg), hypovolemic (with dehydration), edematous (with edema), due to renal failure and hyperglycemic (fictitious) hyponatremia.

The frequency distribution of serum sodium concentration in 227 study children was analysed.Hyponatremia serum sodium <135mEq/L was present in 80 (35.2%) children, while severe hyponatremia (serum sodium ≤125 mEq/ L) was found in 5 chidren (6.25%). Amongst those with severe hyponatremia all the 5 children were in the serum sodium ranged between 121-125 mEq/,none of them in the sodium range <121mEq/L.

Among patients with hyponatremia, symptomatic hyponatremia was found in 4 children. In children with seizures,around 18 patients inaddition to those 4 patients who were symptomatic were given 3% NACL correction.The commonest cause of hyponatremia is cerebral salt wasting and Syndrome of Inappropite ADH secretion.

Among patients with hyponatremia, 9 patients (11.25%) received DNS and 71 patients (88.75%) received ½ DNS. This difference had no statistical significance despite giving this.

Among 25 patients with hyponatremia who died,16 patients (64%) had with seizures died 9 patients (36%) with seizures survived.

Studies on the mechanism of euvolemic hyponatremia have emphasized a role of syndrome of inappropriate ADH secretion (SIADH) in these patients. By conventional criteria, almost all of our patients with euvolemic hyponatremia could be plasma osmolality, high urine osmolality and normal renal function. Inappropriately higher concentration of plasma vasopressin than that expected for the degree of hypo-osmolality has been demonstrated in association with euvolemic hyponatremia in children with meningitis, asthma and hospitalized adults.

Thus, ADH mediated renal salt loss and water retention could be the cause of hyponatremia in such patients. However, severe hyponatremia may occur in association with eSIADH without unusual loss of sodium from the body or

dilution of the plasma sodium. Evidence supporting redistribution and accumulation of sodium within the cells has also been presented.

Recently, Hannon and Boston have shown significant intracellular shifts of sodium chloride and water in septic animals. They suggested that in these animals hyponatremia and hypo-osmolality was caused by a combination of intracellular shift of sodium and water, and dilution of extracellular space probably as a result ofclassified as having SIADH as they had normal hydration, low plasma osmolality

Data from our ongoing research suggest a significant increase in RBC sodium in the presence of hyponatremia in septicemic children. Further studies are needed to clarify the mechanism responsible for hyponatremia in acute infections.

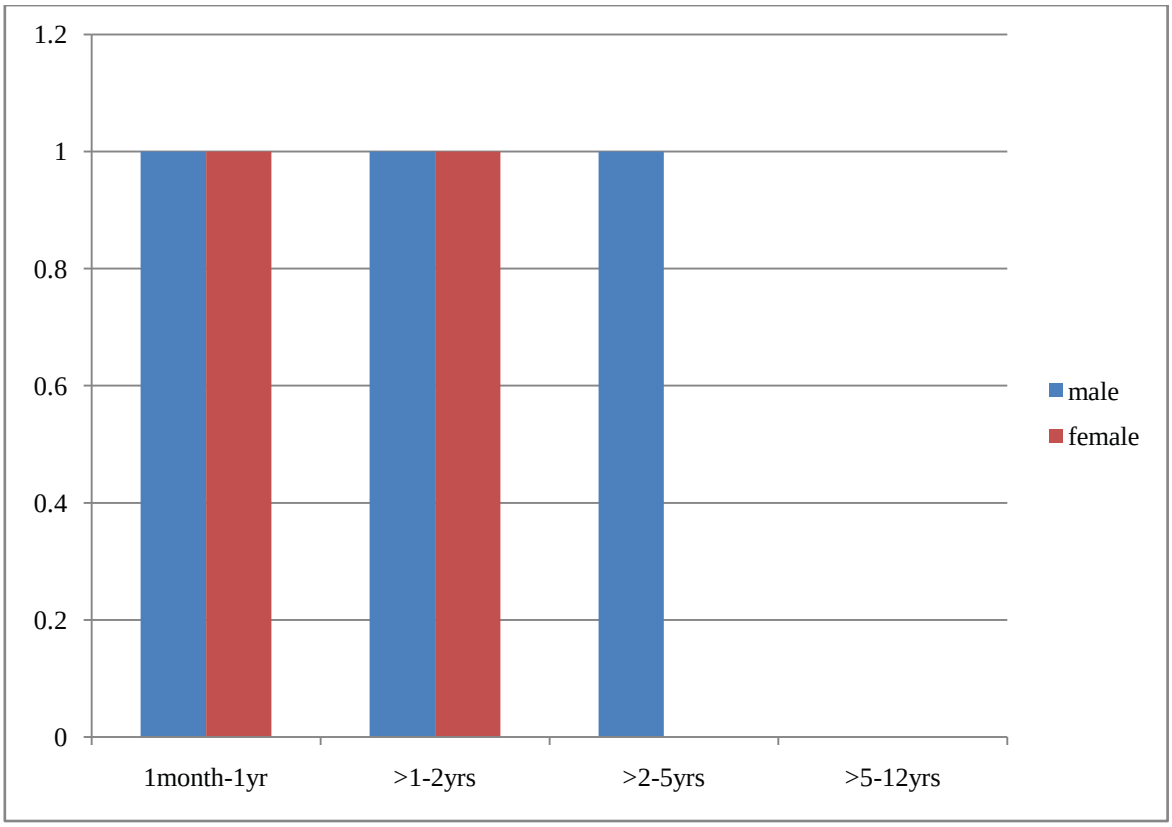


Figure no.8, Sex wise distribution of hypernatremia :

Among 5 cases of Hypernatremia,1 case occurred in both male and female between 1month -1yr and >1-2yrs.There were no case of hypernatremia over the age group of 5-12yrs.out of 5 patients with hypernatremia,3 patients were males, 2 patients were females that is 60 % were males,40 % were females with male to female ratio1.5:1

Potassium disturbances

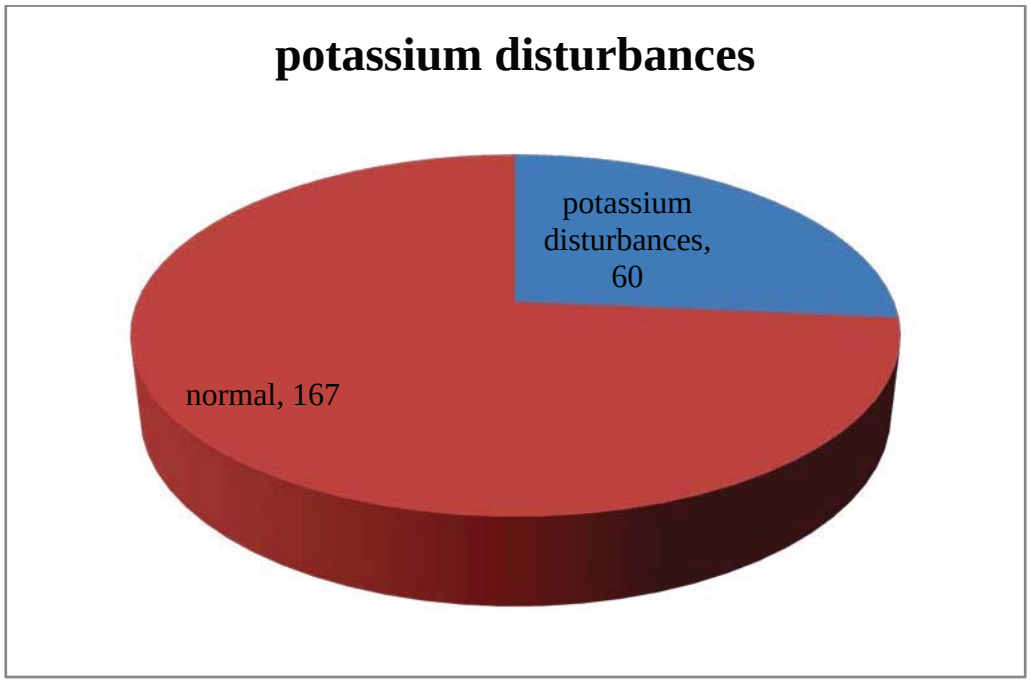


Figure no.10,potassium disturbances and normal electrolyte values

Out of the 227 children,60 children had abnormalities of serum potassium that included both hypokalemia (49 cases)and hyperkalemia(11 cases) accounted for 26.43% of the total.22 patients with mixed electrolyte disturbances that is both sodium and potassium disturbances.

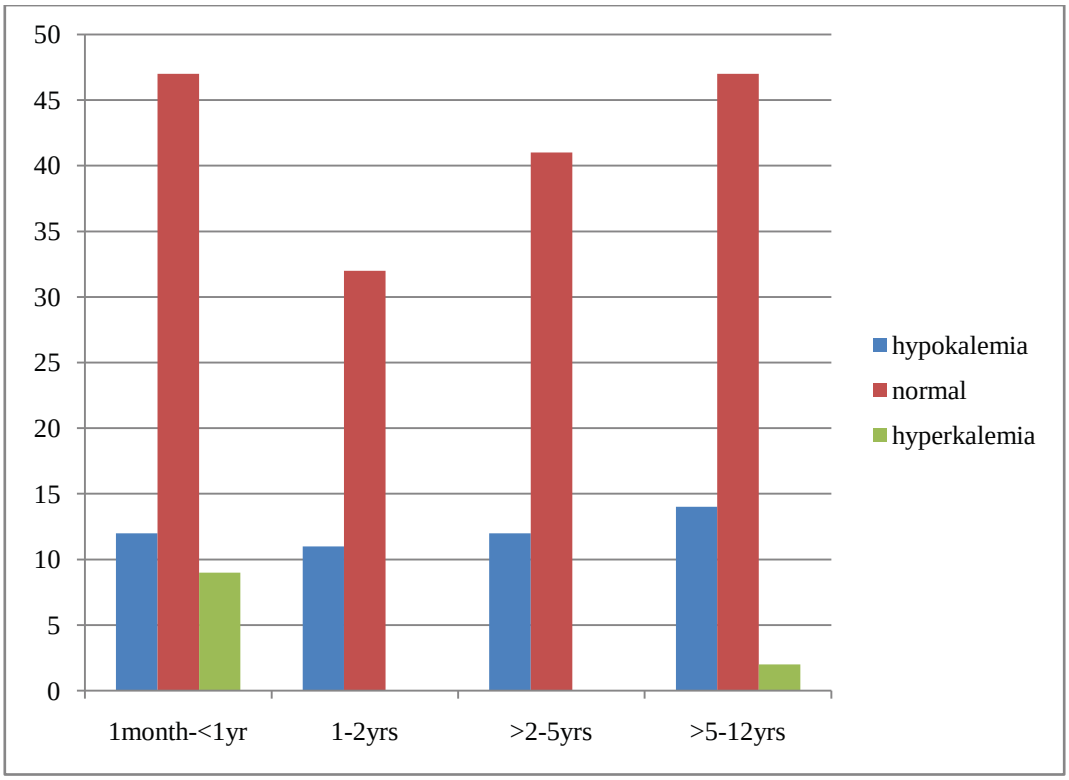


Figure no.11,**Age wise incidence of Potassium disturbances** :

Out of 11 cases of hyperkalemia,Incidence of hyperkalemia is more common among infants between 1month and 1yr of age.the occurrence of hyperkalemia is almost nil in the age group of 1-5yrs.Hereagain higher incidence of hyperkalemia is noted in the age group of 1month to 1year.Incidence of hypokalemia is least among 1-2yr age group.

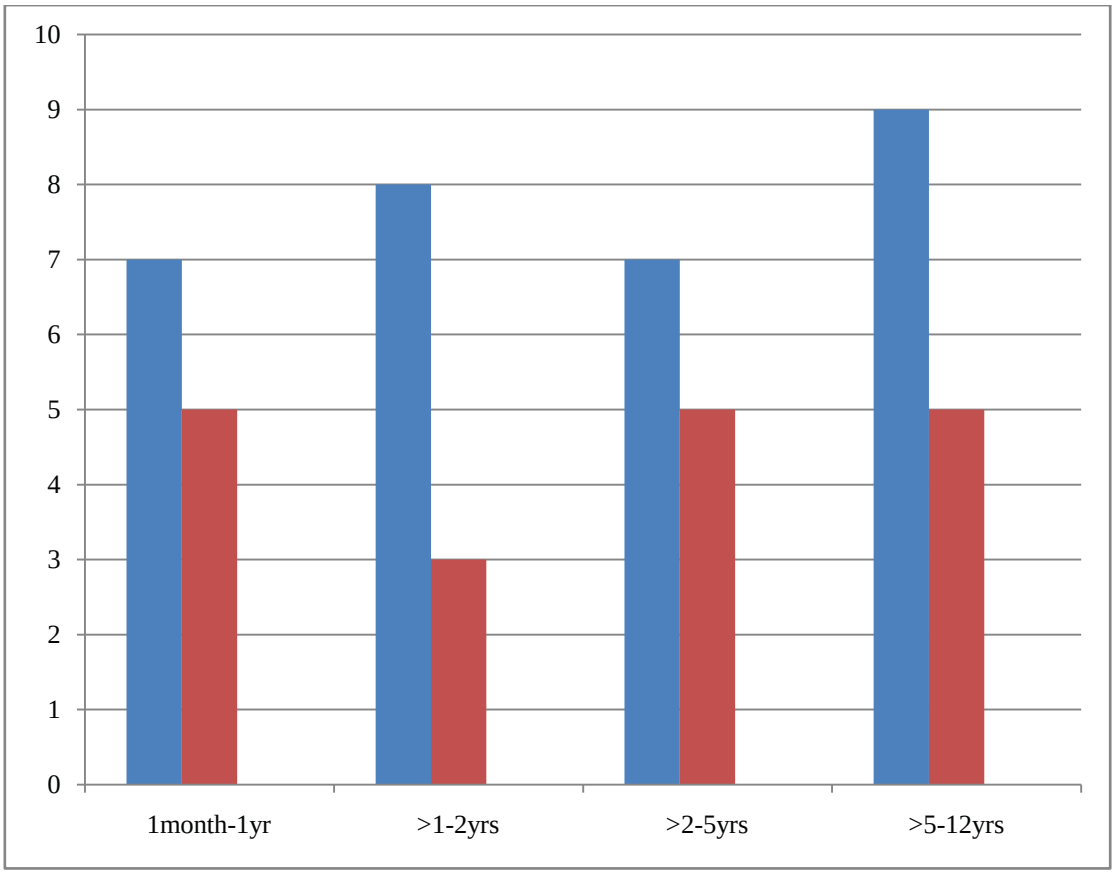


Figure no.12,sexwise distribution of Potassium disturbances

Hypokalemia is more common among male children of age group>5-12yrs that constitutes 18.36 % of hypokalemia caes reported.Among females 5cases each in the age group of 1month-1yr, >2-5yrs and > 5-12yrs. 60 patients developed potassium disturbances that accounted for 26.43% of incidence of total electrolyte disturbances.Data on all those patients, who had hypokalemia documented on at least one occasion during their PICU stay, was analyzed further.

Details of patients regarding their age, sex, weight, diagnoses, clinical course, and outcome were obtained from the records. Details of bio-chemical

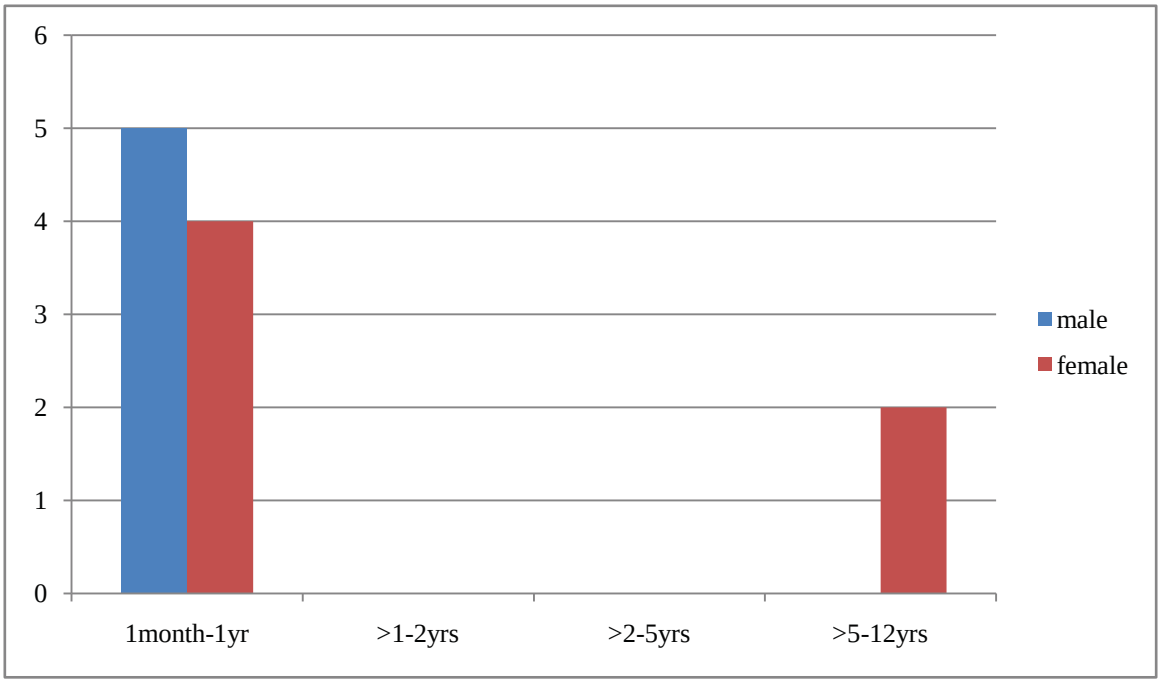
parameters, i.e., electrolytes, acid-base status and renal function parameters, along with ECG and details of treatment were noted.

Hypokalemia was graded as mild if serum potassium ranged between 3.0-3.4 mEq/L, moderate if between 2.0-2.9 mEq/L and severe if <2.0 mEq/L.

Among patients with hypokalemia,28 patients were male children,16 patients were female children,moderate hypokalemia is seen in 3 children in the age group 1-5yrs,none of them were in the severe hypokalemia range.

All the patients received slow intravenous correction in form of increased potassium content of intravenous fluids to 40 to 60 mEq/L.One patient had ECG changes,that received rapid potassium correction.Rapid infusion was given at a rate of 0.3 mEq/kg/h .

Sexwise distribution of hyperkalemia



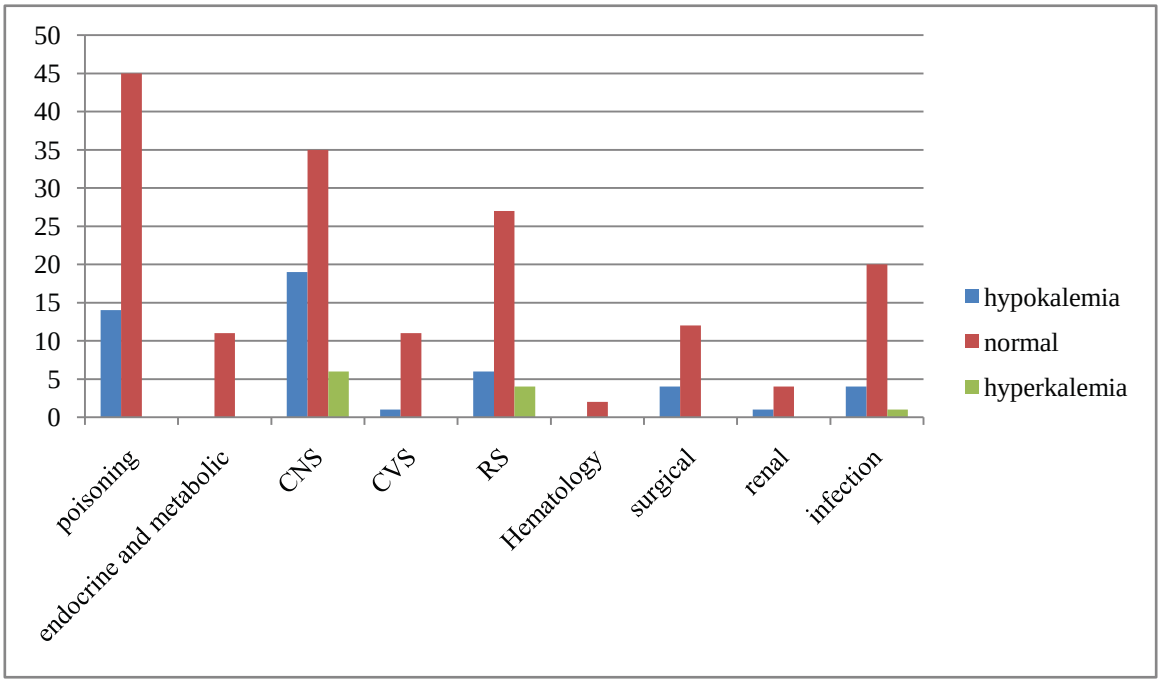
Incidence of hyperkalemia is higher in male children in the age group of 1 month-1 year about 5 cases reported about 4 cases occurred in females of the same age group 1month -1yr.no case was reported in both males and females in the age group of 1-2yrs.There was 2 cases of hyperkalemia occurred in the age group of >5-12yrs among female children and none in the male children.

Among 49 patients with hypokalemia ,31 patients were males,18 were females that is 63.26% were males,36.73% were females with male to female ratio 1.7:1

Among 11 patients with hyperkalemia ,5 patient were male ,6 patients were female

that is 45.45% were males,54.54% were females with male to female ratio1:1.2

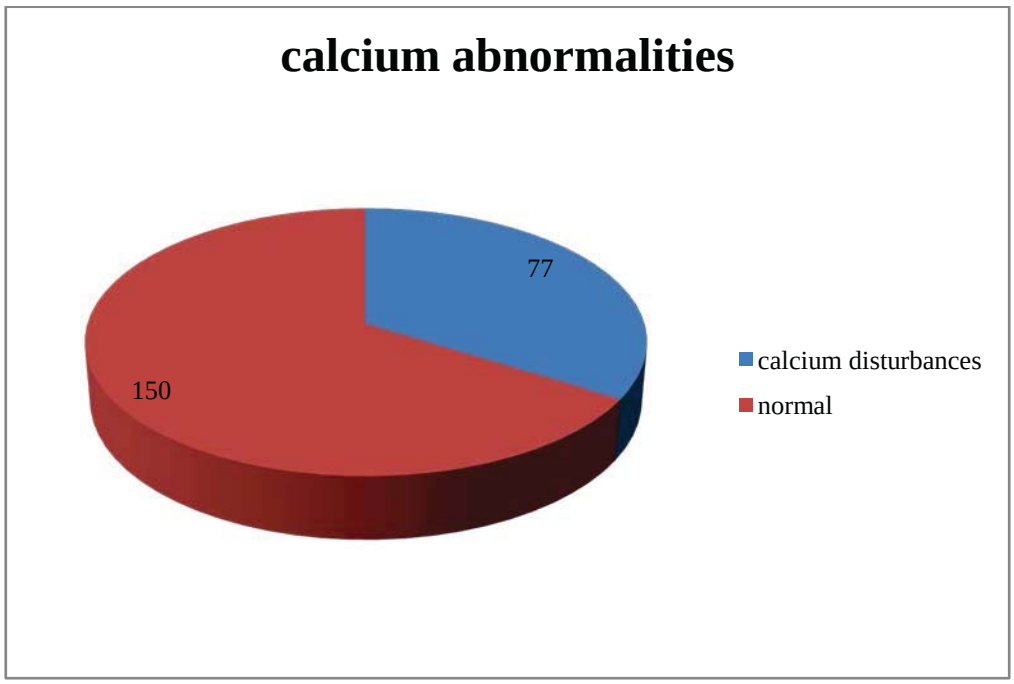
etiology and potassium disturbances



Among 49 patients with hypokalemia, most common etiology was CNS(seizures and acute CNS infection)19 cases contributing 38.78% of hypokalemia cases, followed by poisoning 14 cases 28.57% of hypokalemia.bronchopneumonia 6 cases (12.24%),cardiac renal septicemia each contributing 1 case (2.04%) of hypokalemia.

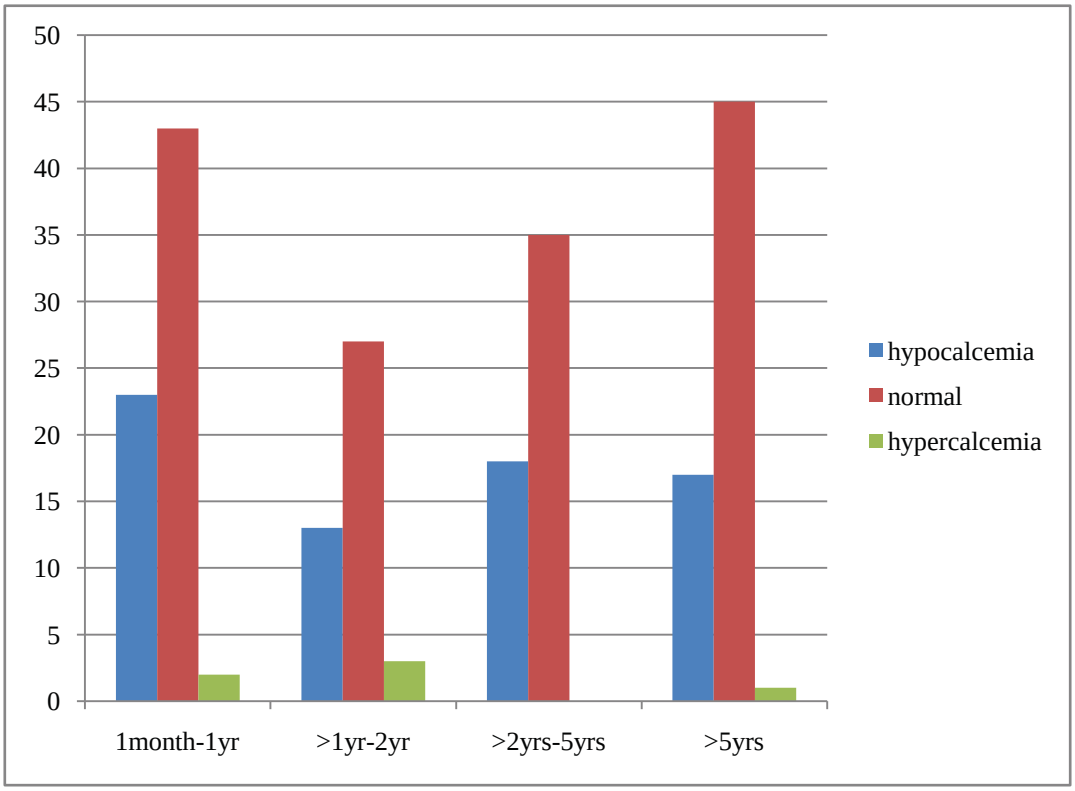
Among 16 patients with hypokalemia 6 patients died due to CNS disorders like seizures and acute CNS infection,4 patient died due to poisoning.

CALCIUM DISTURBANCES



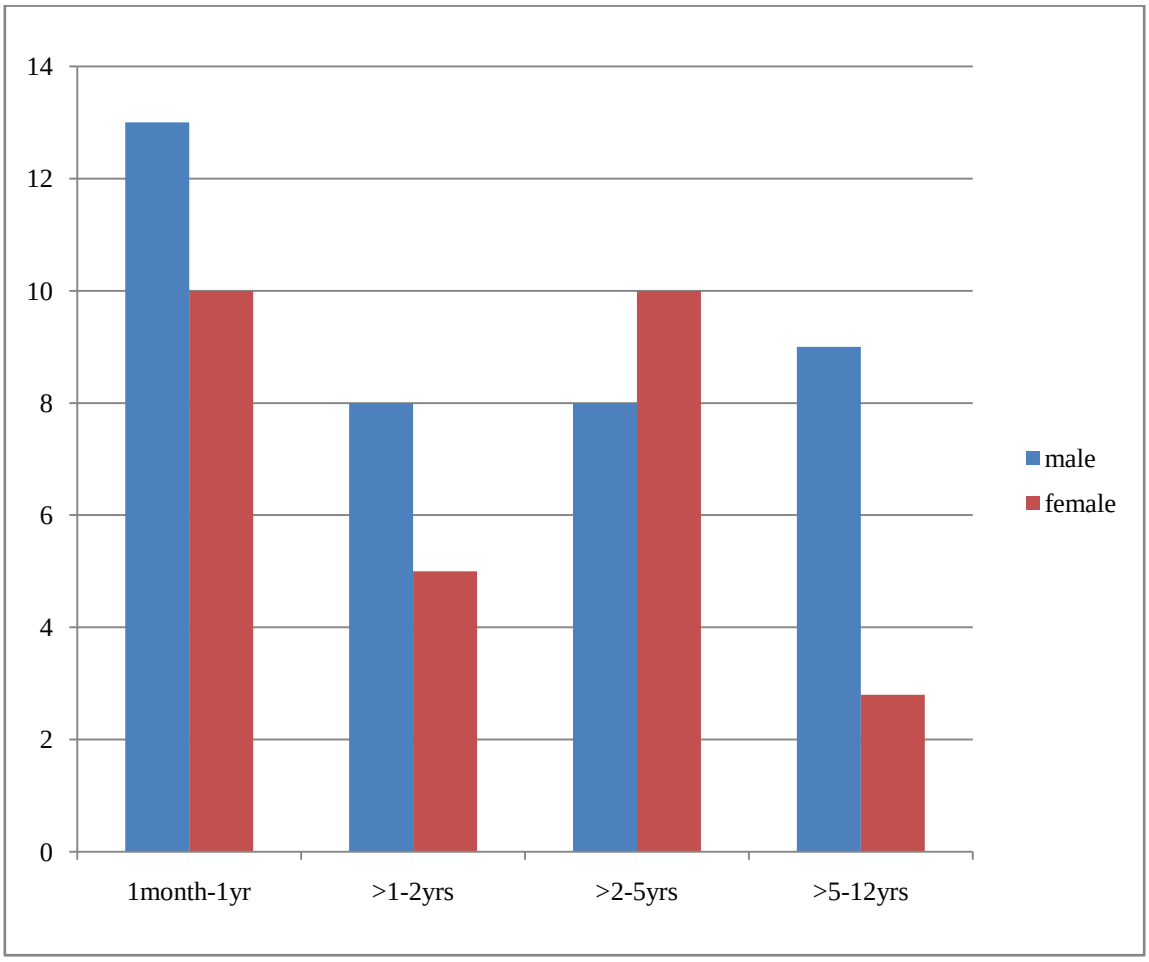
Among the 227 children admitted ,77 children had calcium disturbances,that accounted for 33.92% of total children admitted.

Age wise incidence of Calcium disturbances



It is shown in the above chart that incidence of hypocalcemia is more common in the age group of 1month-1yr.Hypercalemia occurs less commonly and maximum in the age group of 1-2yrs.hypocalcemia incidence is least among 1-2yrs,hypercalcemia is least among 2-5yrs age group.

Sex wise disrtribution of hypocalcemia

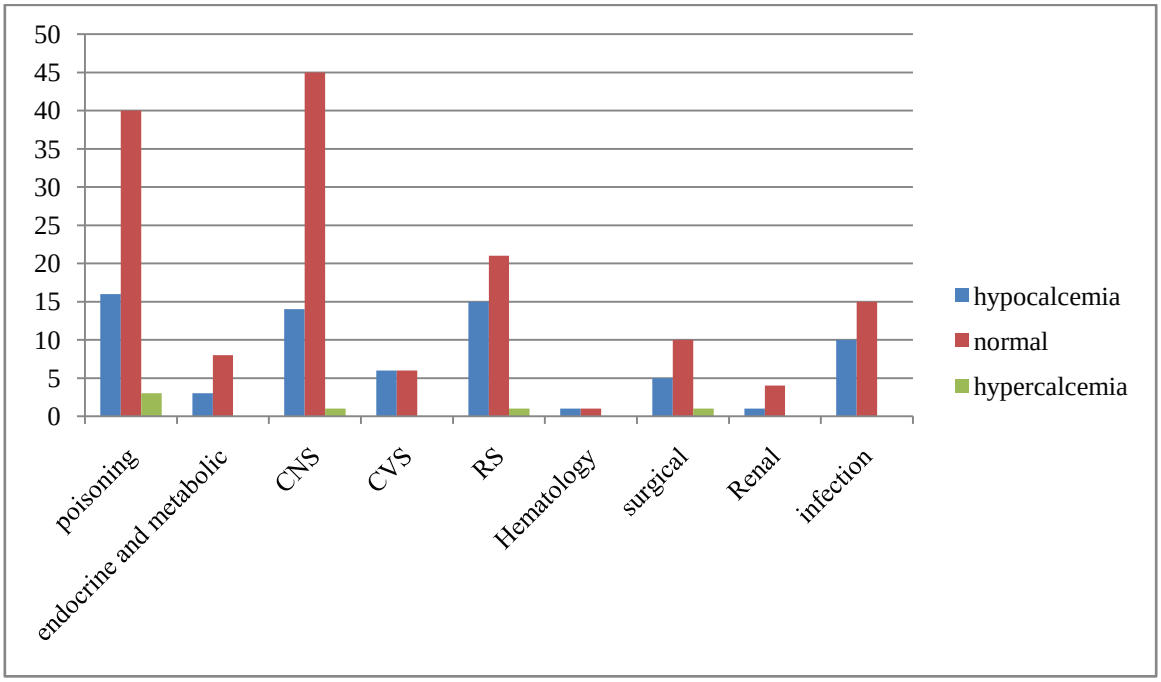


Among 71 patients with hypocalcemia,38 patients were male,33 patients were females that is 53.52% were males,46.47% were females male to female ratio 1.15:1

Maximum number of hypocalcemia 13 cases occurred in the male children of 1month-1yr age group.About 10 cases each occurred in the female children 1month-1yr and again > 2-5yrs age group.Incidence of hypocalcemia is least among female children of age group 5-12yrs.

Among 6 patients with hypercalcemia ,5 patients were males,1 patient were female,83.33% patients were males,16.67% were females,with male to female ratio 5:1

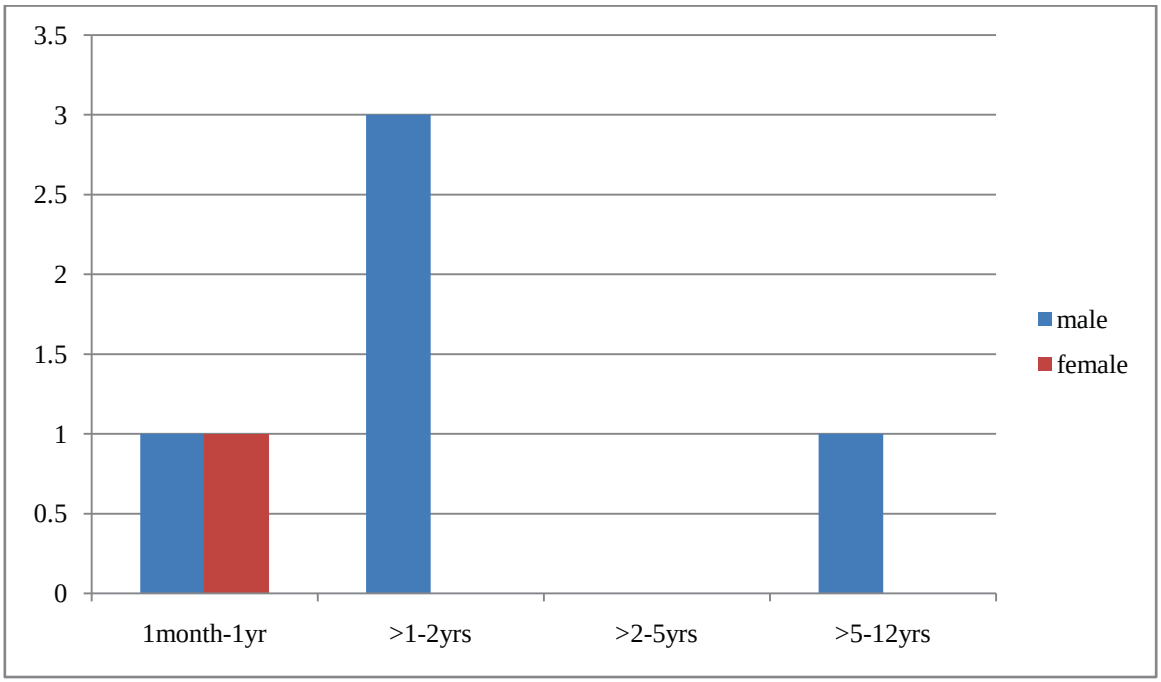
Among 2patients with hypochloremia 1 patient male and 1 female with male to female ratio 1:1



Etiology and calcium disturbances

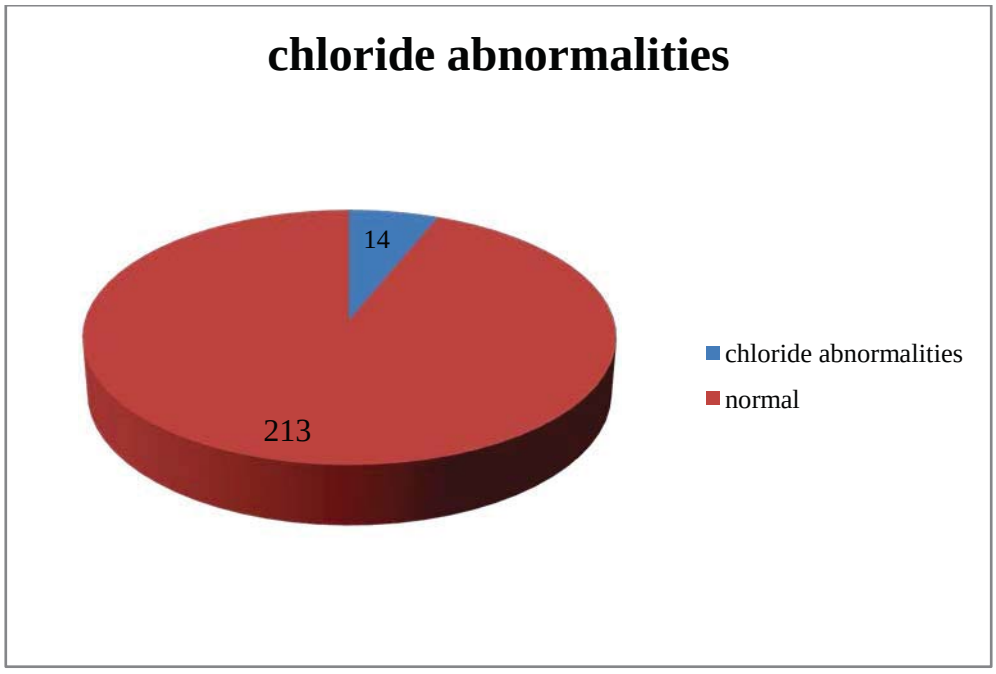
Among patients with hypocalcemia the most frequent cause is established to be poison about 16 20.78% f/b Respiratory like bronchopneumonia 15cases 19.48% f/b CNS 14 cases 18.18%..Among 6 patients with hypercalcemia poison contributed the maximum number 3 (50%) f/b CNS disorders 1(16.67%)

Sexwise distribution of hypercalcemia



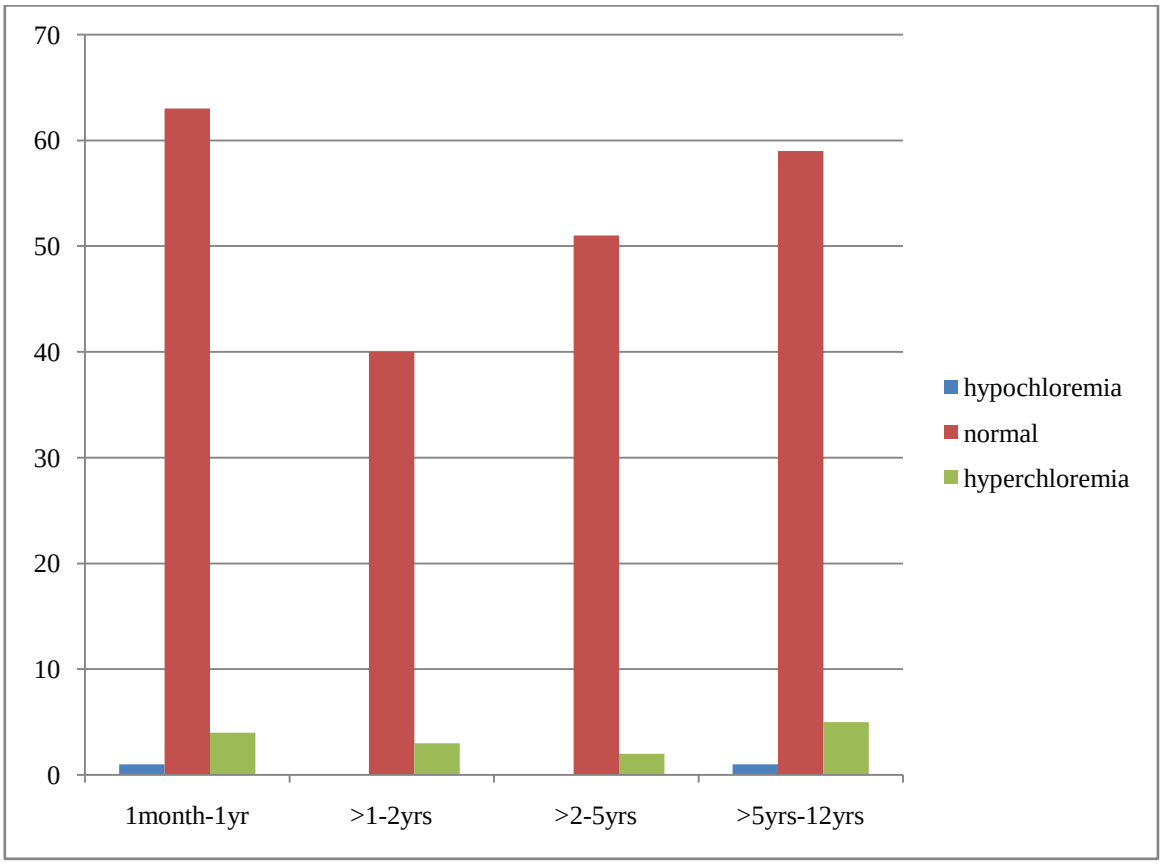
Maximum number of hypercalcemia about 3 cases occurred in male children of 1-2yrs age group with nil cases reported in the 2-5yrs age group.Among female 1 case reported in 1month -1yr age group

Chloride disturbance

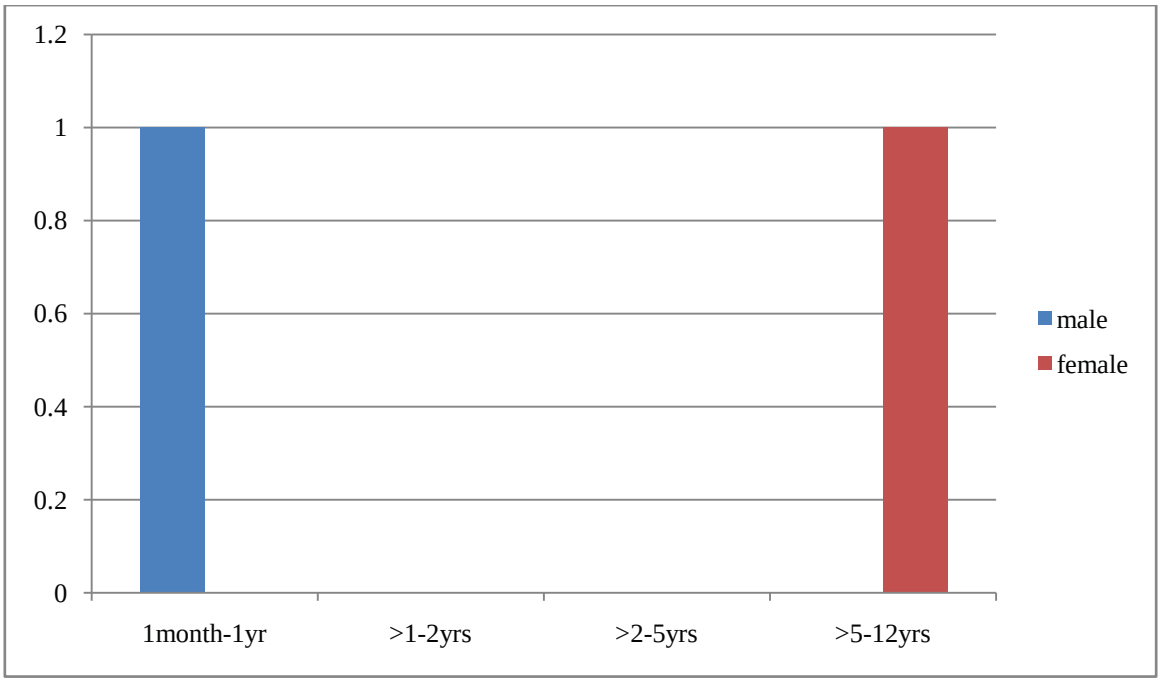


227 children admitted 14 children had chloride abnormalities that accounted for 6.1% of the total.

Age wise incidence of chloride disturbances:

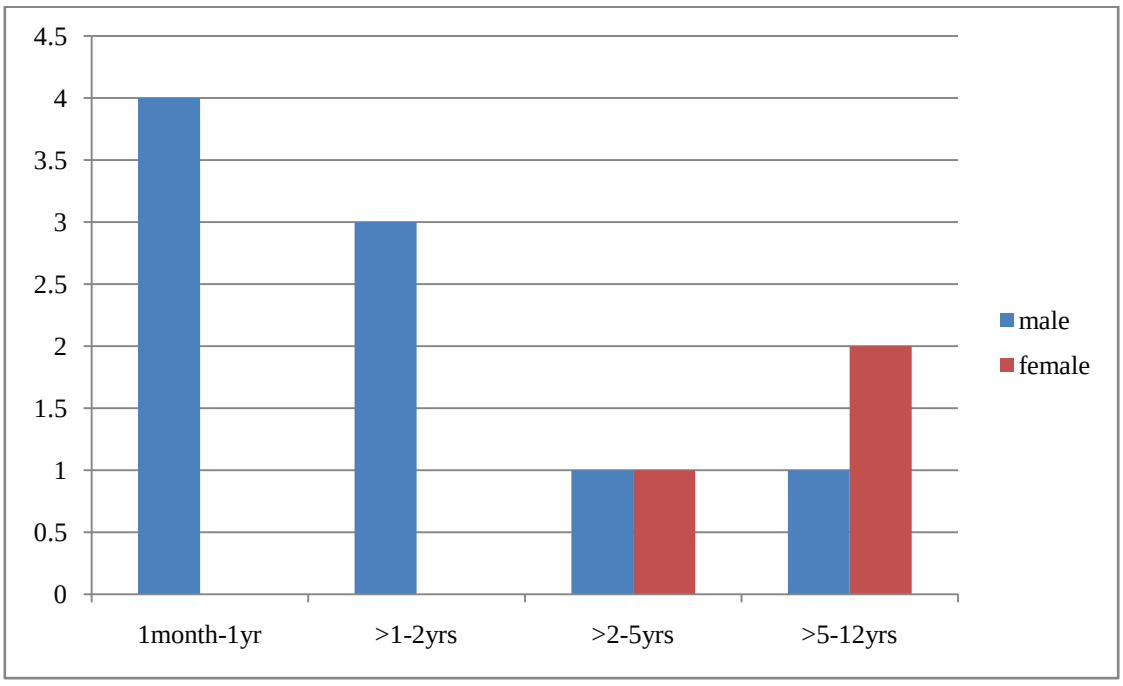


2 case of hypochloremia occurred 1 each in age group of 1month-1yr and >5-12yr.there were 12 hyperchloremia cases maximum in the age group of 1month -1yr about 4 cases.



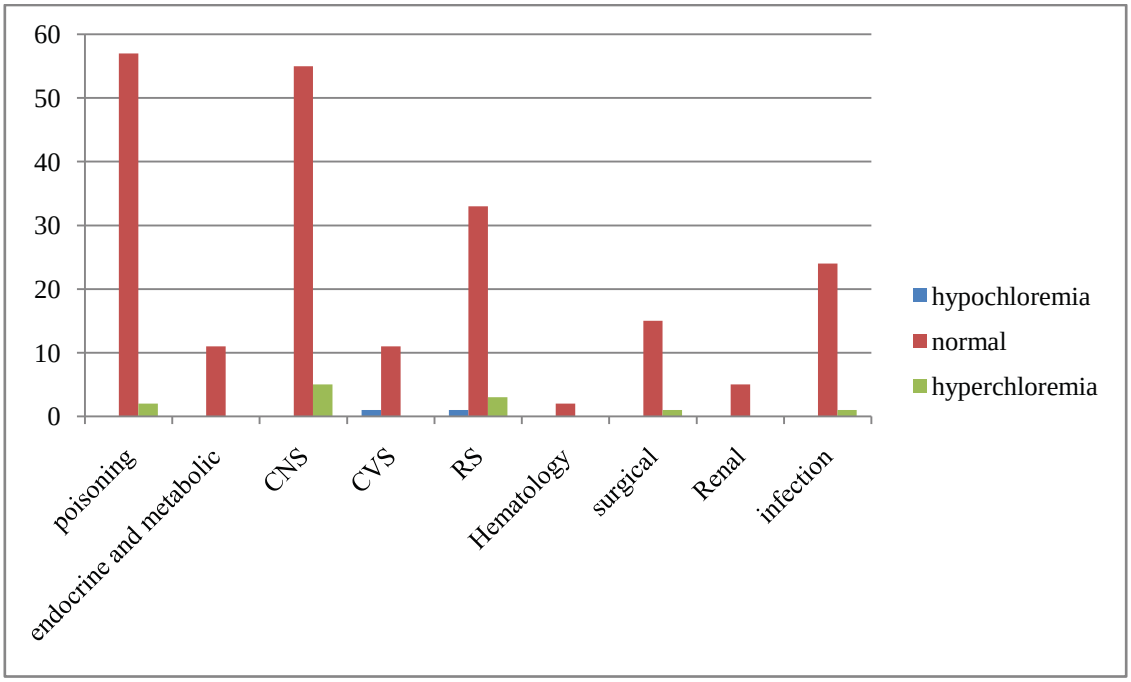
About one case of hypochloremia each of age group 1month-1yr in male and 5-12yrs in female children respectively

Sexwise distribution of hyperchloremia



Among 12 patients with hyperchloremia ,9 patients were male,3 patients were female,75 % were male,25% were females, with male to female ratio of 3 :1

Incidence of hyperchloemia is highest among male children of 1 month-1yr about 4 cases and least in the age group of 2-12yrs about 1 case each.Among female children maximum number of cases occurred in the age group of >5-12yrs.there were no cases reported in the age group of 1month -5yrs agegroup in females.



Maximum number of cases of hyperchloremia was caused by poisoning and CNS disorders like seizure disorder,acute CNS infection

STATISTICAL ANALYSIS

The collected data were analysed with SPSS 16.0 version.To describe about the data, descriptive statistics, frequency analysis, percentage analysis were used for categorical variables and for continuous variables the mean and S.D were used. To find the significant difference between the bivariate samples in independent groups, the independent t test was used. To find the significance in categorical data, Chi-Square test was used. In all the above statistical tools, the probability value of≤0.05 was considered as significant level.

P-Value	Highly Significant at P ≤ .01
P-Value	Significant at P ≤ .05
P-value	No Significant at P >.05

Etiology * Sodium Crosstabulation

			Sodium			
			Hyponatremia	Normal	Hypernatremia	Total
Etiology	Poisoning	Count	17	42	0	59
		% within clinical history	28.80%	71.20%	0.00%	100.00%
	Endocrine and Metabolic	Count	5	5	1	11
		% within clinical history	45.50%	45.50%	9.10%	100.00%
	CNS	Count	25	34	1	60

		% within clinical history	41.70%	56.70%	1.70%	100.00%
	CVS	Count	6	6	0	12
		% within clinical history	50.00%	50.00%	0.00%	100.00%
	RS	Count	11	25	1	37
		% within clinical history	29.70%	67.60%	2.70%	100.00%
	Hematology	Count	2	0	0	2
		% within clinical history	100.00%	0.00%	0.00%	100.00%
	Surgical	Count	6	10	0	16
		% within clinical history	37.50%	62.50%	0.00%	100.00%
	Renal	Count	2	2	1	5
		% within clinical history	40.00%	40.00%	20.00%	100.00%
	Infection	Count	6	18	1	25
		% within clinical history	24.00%	72.00%	4.00%	100.00%

Table no.2,etiology and sodium disturbances

31.25 % of hyponatremia due to poison and statistical analysis done using Pearson chi square test showed no statistical significance between the clinical history of poison and development of hyponatremia.

Etiology * Potassium Crosstabulation						
			Potassium			
			Hypokalemia	Normal	Hyperkalemia	Total
Etiology	Poisoning	Count	14	45	0	59
		% within clinical history	23.70%	76.30%	0.00%	100.00%
	Endocrine and Metabolic	Count	0	11	0	11
		% within clinical history	0.00%	100.00%	0.00%	100.00%
	CNS	Count	19	35	6	60
		% within clinical history	31.70%	58.30%	10.00%	100.00%
	CVS	Count	1	11	0	12
		% within clinical history	8.30%	91.70%	0.00%	100.00%
	RS	Count	6	27	4	37
		% within clinical history	16.20%	73.00%	10.80%	100.00%
	Hematology	Count	0	2	0	2
		% within clinical	0.00%	100.00%	0.00%	100.00%

		history				
	Surgical	Count	4	12	0	16
		% within clinical history	25.00%	75.00%	0.00%	100.00%
	Renal	Count	1	4	0	5
		% within clinical history	20.00%	80.00%	0.00%	100.00%
	Infection	Count	4	20	1	25
		% within clinical history	16.00%	80.00%	4.00%	100.00%

Table no.3,etiology and potassium disturbances

38.75 % of hypokalemia is due to CNS disorders and 54.54% of hyperkalemia is due to CNS disorders this again no statistical significance p value >.05 (0.120) is noted

Etiology * Calcium Crosstabulation						
			Calcium			
			Hypocalcemia	Normal	Hypercalcemia	Total
Etiology	Poisoning	Count	16	40	3	59
		% within clinical history	27.10%	67.80%	5.10%	100.00%
	Endocrine and Metabolic	Count	3	8	0	11
		% within clinical history	27.30%	72.70%	0.00%	100.00%
	CNS	Count	14	45	1	60

		% within clinical history	23.30%	75.00%	1.70%	100.00 %
	CVS	Count	6	6	0	12
		% within clinical history	50.00%	50.00%	0.00%	100.00 %
	RS	Count	15	21	1	37
		% within clinical history	40.50%	56.80%	2.70%	100.00 %
	Hematology	Count	1	1	0	2
		% within clinical history	50.00%	50.00%	0.00%	100.00 %
	Surgical	Count	5	10	1	16
		% within clinical history	31.30%	62.50%	6.30%	100.00 %
	Renal	Count	1	4	0	5
		% within clinical history	20.00%	80.00%	0.00%	100.00 %
	Infection	Count	10	15	0	25
		% within clinical history	40.00%	60.00%	0.00%	100.00 %
Total		Count	71	150	6	227
		% within clinical history	31.30%	66.10%	2.60%	100.00 %

Table no.4,etiology and calcium disturbances

22.53 % children who had poisoning developed hypocalcemia,50% with poisoning developed hypercalcemia.here again the statistical association if found to be not statistically significant

Etiology * Chloride Crosstabulation						
			Chloride			
			Hypochloremia	Normal	Hyperchloremia	Total
clinical history	Poisoning	Count	0	57	2	59
		% within clinical history	0.00%	96.60%	3.40%	100.00%
	Endocrine and Metabolic	Count	0	11	0	11
		% within clinical history	0.00%	100.00%	0.00%	100.00%
	CNS	Count	0	55	5	60
		% within clinical history	0.00%	91.70%	8.30%	100.00%
	CVS	Count	1	11	0	12
		% within clinical history	8.30%	91.70%	0.00%	100.00%
	RS	Count	1	33	3	37
		% within clinical history	2.70%	89.20%	8.10%	100.00%
	Hematolo	Count	0	2	0	2

	gy	% within clinical history	0.00%	100.00%	0.00%	100.00%
	Surgical	Count	0	15	1	16
		% within clinical history	0.00%	93.80%	6.30%	100.00%
	Renal	Count	0	5	0	5
		% within clinical history	0.00%	100.00%	0.00%	100.00%
	Infection	Count	0	24	1	25
		% within clinical history	0.00%	96.00%	4.00%	100.00%

Table no.5,etiology and chloride disturbances

Cardiovascular and respiratory problems contributes each to about 50% of hypochloremia,where as CNS disorders 41.66% and Respiratory system 25% contributed to maximum number of hyperchloremia was not statistically significant

Etiology Outcome Crosstabulation					
			Outcome		
			Death	Improved	Total
clinical history	Poisoning	Count	5	54	59
		% within clinical history	8.50%	91.50%	100.00%
	Endocrine and Metabolic	Count	6	5	11
		% within clinical history	54.50%	45.50%	100.00%
	CNS	Count	16	44	60
		% within clinical history	26.70%	73.30%	100.00%
	CVS	Count	6	6	12
		% within clinical history	50.00%	50.00%	100.00%
	RS	Count	12	25	37
		% within clinical history	32.40%	67.60%	100.00%
	Hematology	Count	0	2	2
		% within clinical history	0.00%	100.00%	100.00%
	Surgical	Count	4	12	16
		% within clinical history	25.00%	75.00%	100.00%
	Renal	Count	2	3	5
		% within clinical history	40.00%	60.00%	100.00%
	Infection	Count	4	21	25

		% within clinical history	16.00%	84.00%	100.00%
Total		Count	55	172	227
		% within clinical history	24.20%	75.80%	100.00%

Table no.6,etiology and outcome

29 % of patients with CNS disorders died f/b 21.81 % of Respiratory problem died, results were analysed using Pearson chisquare test found to be not statistically significant.

Hospital Procedure * Sodium Crosstabulation						
			Sodium			
			Hyponatremia	Normal	Hypernatremia	Total
Hospital Procedure	DNS	Count	9	10	0	19
		% within Hospital Procedure	47.40%	52.60%	0.00%	100.00%
	Half DNS	Count	71	132	5	208
		% within Hospital Procedure	34.10%	63.50%	2.40%	100.00%
Total		Count	80	142	5	227
		% within Hospital Procedure	35.20%	62.60%	2.20%	100.00%

Table no.7 ,maintenance fluids and sodium disturbances

The result showed higher incidence of hyponatremia in those children who received ½ DNS with statistical analysis found p value of 0.1 that is not statistically significant.

Hospital Procedure * Outcome Crosstabulation					
			Outcome		
			Death	Improved	Total
Hospital Procedure	DNS	Count	5	14	19
		% within Hospital Procedure	26.30%	73.70%	100.00%
	Half DNS	Count	50	158	208
		% within Hospital Procedure	24.00%	76.00%	100.00%
Total		Count	55	172	227
		% within Hospital Procedure	24.20%	75.80%	100.00%

Table no.8,maintenance fluid and outcome pattern

Here again there is no significant correlation between use of ½ DNS and DNS with reference to outcome of the children

Hyponatremia more common in the age group of 1month to 1yr with p value of 0.1,not statistically significant

11 male children contributed about 13.75% of hyponatremia.

10 female children constitute 12.5 % of hyponatremia in 5-12yrs age group.

1-2 year age group incidence of hyperchloremia is 33.33 % in male children

Sodium * Outcome Crosstabulation					
			Outcome		
			Death	Improved	Total
Sodium	Hyponatremia	Count	25	55	80
		% within Outcome	45.50%	32.00%	35.20%
	Normal	Count	29	113	142
		% within Outcome	52.70%	65.70%	62.60%
	Hypernatremia	Count	1	4	5
		% within Outcome	1.80%	2.30%	2.20%
Total		Count	55	172	227
		% within Outcome	100.00%	100.00%	100.00%

Table.no.9,sodium disturbances and outcome pattern

To determine the significance for the above categorical data, Pearson chi-square statistical test was employed. The P-value was estimated to be 0.579 (>0.05). Hence the percentage of deaths or discharges among the patients with sodium disturbances of the study population has not been found statistically significant.

Potassium * Outcome Crosstabulation					
			Outcome		
			Death	Improved	Total
Potassium	Hypokalemia	Count	16	33	49
		% within Outcome	29.10%	19.20%	21.60%
	Normal	Count	38	129	167
		% within Outcome	69.10%	75.00%	73.60%
	Hyperkalemia	Count	1	10	11
		% within Outcome	1.80%	5.80%	4.80%
Total		Count	55	172	227
		% within Outcome	100.00%	100.00%	100.00%

Table no.10,potassium disturbances and outcome pattern

To determine the significance for the above categorical data, Pearson chi-square test was employed. We then arrived at a P-value of 0.146 (>0.05). Hence the percentage of deaths or discharges among patients with potassium disturbances has not been found statistically significant.

Calcium * Outcome Crosstabulation					
			Outcome		
			Death	Improved	Total
Calcium	Hypocalcemia	Count	23	48	71
		% within Outcome	41.80%	27.90%	31.30%
	Normal	Count	31	119	150
		% within Outcome	56.40%	69.20%	66.10%
	Hypercalcemia	Count	1	5	6
		% within Outcome	1.80%	2.90%	2.60%
Total		Count	55	172	227
		% within Outcome	100.00%	100.00%	100.00%

Table no.11,calcium disturbances and outcome pattern

There is no significant difference between hypocalcemia and death in these children

Chloride * Outcome Crosstabulation					
			Outcome		Total
			Death	Improved	
Chloride	Hypochloremia	Count	0	2	2
		% within Outcome	0.00%	1.20%	0.90%
	Normal	Count	54	159	213
		% within Outcome	98.20%	92.40%	93.80%
	Hyperchloremia	Count	1	11	12
		% within Outcome	1.80%	6.40%	5.30%
Total		Count	55	172	227
		% within Outcome	100.00%	100.00%	100.00%

Table no.12,chloride disturbances and outcome pattern

This showed no significant differences in outcome between the chloride abnormalities

Etiology * Outcome Crosstabulation					
			Outcome		Total
			Death	Improved	
clinical history	Poisoning	Count	5	54	59
		% within Outcome	9.10%	31.40%	26.00%
	Endocrine and Metabolic	Count	6	5	11
		% within Outcome	10.90%	2.90%	4.80%
	CNS	Count	16	44	60
		% within Outcome	29.10%	25.60%	26.40%
	CVS	Count	6	6	12
		% within Outcome	10.90%	3.50%	5.30%
	RS	Count	12	25	37
		% within Outcome	21.80%	14.50%	16.30%
	Hematology	Count	0	2	2
		% within Outcome	0.00%	1.20%	0.90%
	Surgical	Count	4	12	16
		% within Outcome	7.30%	7.00%	7.00%
	Renal	Count	2	3	5

		% within Outcome	3.60%	1.70%	2.20%
	Infection	Count	4	21	25
		% within Outcome	7.30%	12.20%	11.00%
Total		Count	55	172	227
		% within Outcome	100.00%	100.00%	100.00%

Table no.13,etiology and outcome pattern

Incidence of death among specific etiology showed P value of 0.12 and found to be not statistically significant between actual cause of admission and death.

Discussion

Profile of serum electrolyte disturbances:

This prospective study evaluated the frequency, clinical characteristics and causes of hyponatremia (serum sodium < 135 mEq/L) in 227 children upto 12 years of age, who were brought for emergency care, and needed hospitalization.Hyponatremia was found in The frequency of hyponatremia in children with pneumonia was 13.75% while in meningitis/encephalitis it was 31.25%. This is similar to that reported by Shann and Germer and Gonzalez et al. The euvolemic type accounted for 80-90% of hyponatremia in these children as also in children with septicemia. Similar observations were made by Gonzalez et al. in children with acute infections.

Apparently, hyponatremia occurs frequently without any major alterations inextracellular fluid volume in children with infectious diseases requiring hospitalization, and should therefore be looked for actively in children with these diagnostic entities, and managed appropriately.

Similar observations were found in another study by SVS.Prasad,Sunit singh and K.S.Chugh in PGIMER Chandigarh, The study has shown that hyponatremia occurs

frequently in sick children requiring emergency care, especially in summer months, and should receive appropriate attention in the management plan.

Hypernatremia:

Out of 227 patients studied ,hypernatremia was seen in 5 patients,with seizures,bronchopneumonia,AGN,septicemia,metabolic each contributing 1 case.most of them were managed conservatively.

1 case died of hypernatremia,due to bronchopneumonia.none of the patient with hypernatremia were symptomatic.

Similar observation were noted in a study by Michael et al, journal of critical care (2013) hypernatremia was independently associated with a 40% increase in risk for hospital mortality and 28% increase in PICU length of stay.

Potassium disturbances :

21 patients developed potassium disturbances of which 12 had hypokalemia and 9 patients had hyperkalemia hereagain the most common age group being 1month-1year this accounted for 35 % of potassium disturbances.

Similar features were seen in another study by S.Singhi and A.Marudhukar PGIMER

Hypokalemia is a common problem among PICU patients. Early detection through regular monitoring and rapid correction may help in improving the outcome. suggest that regular monitoring, of serum potassium, along with supportive ECG monitoring, is useful for proper management of PICU patients. Rapid correction of hypokalemia may be life saving.

Hyperkalemia :

Hyperkalemia was found in 11 patients out of 227 patients.patients were grouped into mild (potassium 5.5-6.5mEq/L) moderate (potassium >6.5-7.5mEq/L) and severe (potassium >7.5 mEq/L) hyperkalemia.Among mild hyperkalemia 8 children were in the age group of 1month-1 year, 2children (both female) in the age group of >5-12yrs and 1 children with severe hyperkalemia in the 1month-1yr age group.

ECG changes was seen in 1 children,calcium gluconate correction was given for this child.these 11 patients with hyperkalemia were asymptomatic during the course.

Most common etiology of hyperkalemia was seizures about 6 cases(54.54%) followed by bronchopneumonia 4 caess(36.36%),renal failure 1case(9%).

About 1 case died of hyperkalemia etiology being seizure disorder. Rest of 11 patients were improved.

Similar study were seen An et al .critical care 2012,korea where they showed severe hyperkalemia occurred in various medical condition .the mortality rate is especially high in patients with severe underlying disease,coexisting medical condition and those with normal baseline renal function.

Hypocalcemia :

25 patients developed calcium disturbances of which 23 had hypocalcemia , 2 patients developed hypercalcemia, this accounted for 32.46% of calcium disturbances

71 patients had hypocalcemia (31.3%).most common age group being 1month-1yr.symptomatic hypocalcemia were found in 6 patients,calcium gluconate correction was given in 6 patients.Most common cause of hypocalcemia was poisoning 16 cases 2.53%),bronchopneumonia 15 cases (21.12%) , seizures14cases (19.71%), septicemia10 cases (14.08%).

Mortality of 23 cases again the most common age group being 1month-1yr,maximum mortality among poisoning cases.

Similar observation were seen in another study by Padhi.R et al hypocalcemia is a frequent finding in critically ill patients.patients with ionized hypocalcemia had a longer ICU stay,longer mechanical ventilation days and a higher mortality rates than those with normal calcium levels.

Hypercalcemia :

6 out of 227 children had hypercalcemia.poisoning 3 cases,with seizures,bronchopneumonia,surgical each contributing 1 case.maximum number of cases occurred in the age group of >1-2yrs.None of them were symptomatic.1 case died due to bronchopneumonia.

Another study done by Jerry M.Earll,Kurtzman showed that hypercalcemia has been associated with reversible hypertension.their study confirms the importance of ionic calcium concentrations on peripheral vascular tone .

Hypochloremia :

14 patients developed chloride disturbances,this accounted for 6.2% of incidence of chloride disturbances

2 out of 227 patients had hypochloremia bronchopneumonia and arrhythmia each contributed 1 case and none of them were symptomatic.no mortality were found in these children.1 case in male occurred in age group of 1month-1yr,another case in female in the age group of >5-12yrs.

Hyperchloremia :

Hyperchloremia were found in 12 patients.seizures 5,bronchopneumonia 3,poisoning 2,surgical 1,septicemia 1.4 children were in the age group of 1 month-1yr,3 children each in the age group of >1-2yrs and >5-12yrs each.1 child with hyperchloremia died etiology being seizures.here again none of them were symptomatic.

POISONING kerosene poisoning is found in 49 patients that accounted for 47.11 %of poisoning cases.

77 patients developed calcium disturbances that accounted for 33.9% incidence of serum calcium disturbances.

The knowledge of prevalence of electrolyte disturbances in a hospital is the most essential and the foremost in planning appropriate preventive strategies in that particular hospital. This information also stays as a baseline value for future comparison of results following any interventions taken in this regard. Thus the value of the effort taken for this part of the study is well acceptable..

But when correlation between the age distribution and the clinical outcome were analysed, the percentage of deaths or discharges among the various age groups of the study population were not found to be of any statistical significance.

The sex distribution of the study population was then correlated with the clinical outcome and statistical analysis done for the same. Once again the percentage of deaths or discharges among the male or female population has not been found to be statistically significant.Out of 55 deaths reported over the period of study,25 pateints had hyponatremia 16 patients had hypokalemia ,23patients had hypocalcemia. 5 cases died had received DNS fluid and 50 cases died received ½ DNS ,these results were not statistically significant.

Conclusion

In conclusion, hyponatremia occurs frequently and should be looked for in all sick children.It is mostly of euvolemic type in almost all the acute infections except diarrhea and should be managed accordingly. Most common electrolyte abnormality observed in the study is hyponatremia.

Hypokalemia is a common electrolyte disturbances among PICU patients. Early detection through regular monitoring and rapid correction may help in improving the outcome.Many of these times it is not associated with any ECG abnormality but still have electrolyte abnormaity,therefore high index of suspicion is needed so that we can prevent hypokalemia and its complications.

In view of the above and absence of consistent relationship between serum potassium concentration and ECG changes,we feel that rapid correction of hypokalemia should be used more often intreatment of moderate and severe hypokalemia even in the absence of ECG abnormality

Recommendations :

Administration of routine maintenence fluids, which are generally hypotonic (such as N/5saline,in 5% glucose),may worsen the hyponatremia.

Although administration of ½ DNS was associated with higher incidence of hyponatremia ,it is not statistically significant.

DNS and isotonic fluids 0.9% NS can be considered for maintenance fluids.

Limitation of the study :

Other maintenance fluid like isolyte P, 5% Dextrose were not studied

Randomization was not done

ABG analysis, Plasma Osmolality were not measured in all patients and were done in selected cases.

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PROFORMA

Name : IP no :

Age : DOA :

Sex : DOD :

Address: No of days of stay :

Informant:

Chief complaints :

1.

2.

H/o of presenting illness:

h/o fever : present or not

Vomiting :

thirst :

Diarrhoea :

Constipation :

Chest retraction:

Weight loss :

- Scanty urination >6hrs : present/ not
- Increased urination :
- Pain abdomen :present/absent/vague
- Muscle cramps : present/absent/vague
- Abd distension :present/absent
- Convulsion :present/absent
- Altered sensorium :present/absent
- Palpitation :
- Abnormal breathing :
- Orthostatic hypotension:
- Treatment h/o :
- Past h/o :

h/o hospitalisation

h/o similar illness

any other

- Birth h/o :
- Dietary h/o :

- Developmental h/o:
- Immn h/o :
- Family h/o :
- Clinical examn on admission
- Anthropometry
- Ht (cm) : Wt (kg) : HC (cm) :
- Nutritional status :
- General physical examination

Anterior fontanelle : flat/depressed/bulge

Shrivelled abdomen :present/absent

Pallor :present/absent

Any other significant findings

- Acidotic breathing : present/absent
- status of hydration :

CARDIOPULMONARY ASSESSMENT

- Airway : stable/unstable/obstructed
- Breathing : RR-

- Nasal flare/grunting/stridor/retraction
- Abdominal/thoracic air entry
- Tidal volume:
- Crepts/wheeze:
- Colour :
- Circulation : HR
- Sounds

Pulse volume :

- cool below thigh/knee/ankle :
- CRT :
- Liver span :
- BP :
- SaO2
- Disability :
- Alert/verbal/pain/unresponsive
- Pupil :
- Eye movements :

- Tone/posture :
- Cardiovascular system :
- Respiratory system :
- Abdomen :
- Central nervous system:

sensorium :alert/irritable/lethargy/unconscious

hypotonia :present/absent

DTR :normal/decreased/increased

- **Physiological status**
- Airway: stable/not stable/obstructed
- Breathing: normal/effortless tachypnoea/respiratory distress/failure/relative bradypnea/apnea
- Circulation:HR N/perfusion normal/tachycardia/relative bradycardia/bradycardia
- Shock/liver span n/increased,cardiogenic shock/BP N/low/high
- Disability :Nil/ALOC/CSE/NCSE/ICP
- **DIAGNOSIS :**

- **TREATMENT:**

- Ventilated/not

- Shock present/absent

- Ionotropes given/not

- Other management

- OUTCOME:

- Death/disability/discharged cured

சுய ஒப்புதல் படிவம்

தீவிர சிகிச்சை பிரிவில் அனுமதிக்கப்படும் குழந்தைகளின் எலக்ட்ரோலைட் தொந்தரவுகளும் மற்றும் எலக்ட்ரோலைட் தொந்தரவுகளால் ஏற்படும் பின் விளைவுகளை கண்டறிதல்

ஆய்வாளர் : ஜெ.பாலாஜி
முதுநிலை பட்டமேற்படிப்பு மாணவர்,
குழந்தை நலத்துறை

வழிகாட்டி: பேராசிரியர் மரு.சாந்தி
குழந்தை நலத்துதறை
அரசு ஸ்டான்லி மருத்துவமனை

பங்கு பெறும் நோயாளியின்
பெயர்: வயது: உள்ளிருப்பு எண்:

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை தீர்க்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது.

நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். ஏந்த காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்ட சிக்கலும் இன்றி இந்த ஆய்வில் இருந்து விலகிக் கொள்ளலாம் என்று அறிந்து கொண்டேன்.

நான் ஆய்வில் இருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவை இல்லை எனவும் அறிந்து கொண்டேன். என்னை பற்றிய தகவல்கள் இரகசியமாக பாதுகாக்கப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையம் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப பயன்படுத்திக்கொள்ளவும் அதனை பிரசுரிக்கவும் முழு மனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். எனக்கு கொடுக்கப்பட்டுள்ள அறிவுரைகளின்படி நடந்து கொள்ளவதுடன் ஆய்வாளருக்கு உண்மையுன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

உடல்நலம் பாதிக்கபட்டாலோ வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ அதனை தெரிவிப்பேன் என்று உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையம்இ சிகிச்சைகளையும் மேற்கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு

நோயாளியின் கையொப்பம் ஆய்வாளரின் கையொப்பம்

(பெயர்)

நோயாளி தகவல் தாள்

தீவிர சிகிச்சை பிரிவில் அனுமதிக்கப்படும் குழந்தைகளின் எலக்ட்ரோலைட் தொந்தரவுகளும் மற்றும் எலக்ட்ரோலைட் தொந்தருவுகளால் ஏற்படும் பின் விளைவுகளை கண்டறிதல்

ஆராய்ச்சியின் நோக்கமும், ஆதாயங்களும்:

எனது ஆராய்ச்சி தீவிர சிகிச்சை பிரிவில் அனுமதிக்கபடும் குழந்கைளின் இரத்தத்தில் பெருவாரியான எலக்ட்ரோலைட் தொந்தரவு எது என்பதை கண்டறிதல் மற்றும் எலக்ட்ரோலைட் தொந்தரவுகளால் ஏற்படும் பின் விளைவுகளை கண்டறிதல்.

ஆய்வுமுறை:

எனது ஆராய்ச்சி தீவிர சிகிச்சை பிரிவில் அனுமதிக்கப்படும் குழந்தைகளின் எலக்ட்ரோலைட் தொந்தரவு அளவு எவ்வளவு என்று அறியப்படும்.

உண்டாகக்கூடிய இடர்கள்:

இந்த ஆய்வில் 2 மில்லி இரத்தம் பரிசோதனைக்கு அனுப்பப்படுகிறது. இதனால் குழந்தைகளுக்கு தீங்கு இலலை.

ஆய்வில் உங்கள் உரிமைகள்:

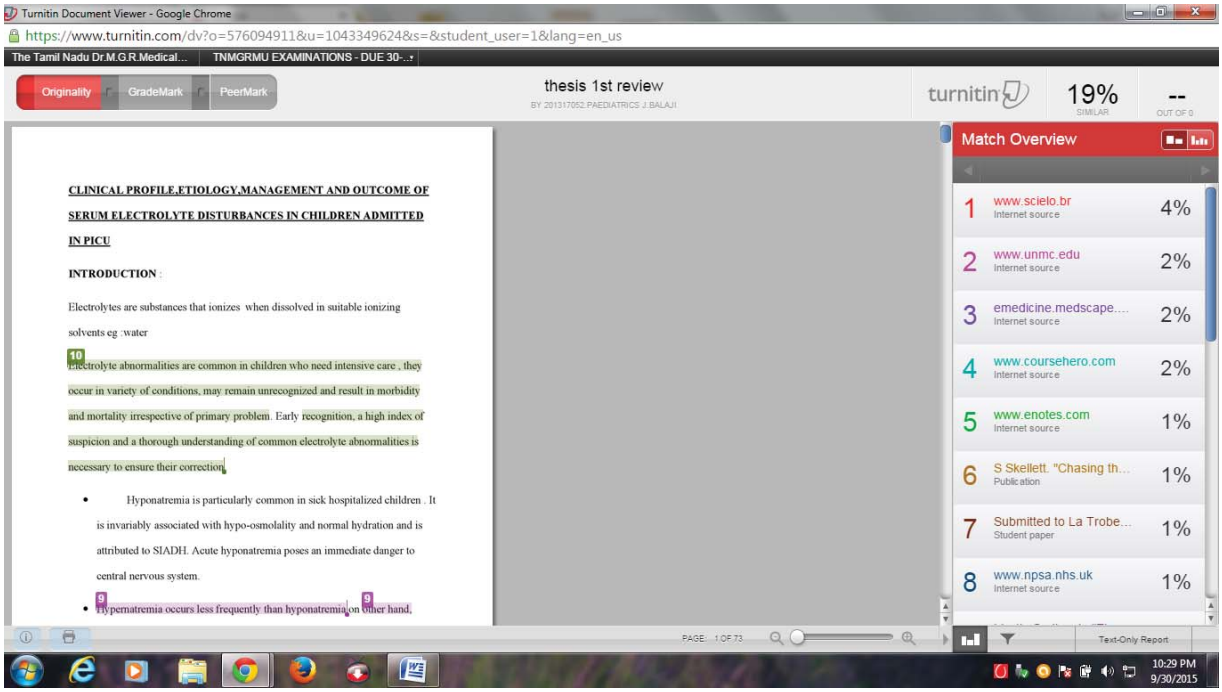
உங்கள் மருத்துவ பதிவேடுகள் அந்தரங்கமாக வைத்துக் கொள்ளப்படும். இந்த ஆய்வின் முடிவுகள் அறிவியல் பத்திரிக்கைகளில் வெளியிடப்படலாம். ஆய்வில் பங்கேற்பது தன்னிச்சையானது மற்றும் காரணங்கள் எதுவும் கூறாமலேயே நீங்கள் எப்போது வேண்டுமென்றாலும் விலகிக் கொள்ளலாம். ஏதேனும் பக்க விளைவுகள் ஏற்பட்டால் முழு சிகிச்சையும் மருத்துவ குழுவினரால் உடனடியாக வழங்கப்படும்

நாள்:

நோயாளியின் கையொப்பம்
(அல்லது இடது பெருவிரல் ரேகை)
(மருத்துவரால் படித்து காட்டப்பட்டது)

ABBREVIATIONS

CNS	- Central nervous system
CVS	- Cardiovascular system
RS	- Respiratory system
SIADH	- Syndrome of inappropriate ADH secretion
ADH	- Antidiuretic hormone
PICU	- Pediatric intensive care unit
DNS	- Dextrose normal saline
ECG	- Electrocardiography
ECHO	- Echocardiogram
NACL	- Sodium chloride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CLINICAL PROFILE,ETIOLOGY,MANAGEMENT AND OUTCOME OF SERUM ELECTROLYTE DISTURBANCES IN CHILDREN ADMITTED IN PICU
INTRODUCTION :
Electrolytes are substances that ionizes when dissolved in suitable ionizing solvents eg :water
Electrolyte abnormalities are common in children who need intensive care , they occur in variety of conditions, may remain unrecognized and result in morbidity and mortality irrespective of primary problem. Early recognition, a high index of suspicion and a thorough understanding of common electrolyte abnormalities is necessary to ensure their correction.
Hyponatremia is particularly common in sick hospitalized children . It is invariably associated with hypo-osmolality and normal hydration and is attributed to SIADH. Acute hyponatremia poses an immediate danger to central nervous system.
Hypernatremia occurs less frequently than hyponatremia on other hand,
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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Clinical profile , Etiology, management and outcome of serum electrolyte disturbances in children admitted in PICU in tertiary care centre.

Principal Investigator : Dr. J Balaji.

Designation : PG MD (Paediatrics)

Department : Department of Paediatrics
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.01.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

K Vasantha

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

case no	gender	age	wt in kg	serum sodium		serum potassium		serum calcium		serum bicarbonate		serum chloride		ABG analysis	urine electrolyte	clinical history	management	outcome
				initial	repeat	initial	repeat	initial	repeat	initial	repeat	initial	repeat					
1	m	7yr	24	137		4.2		9.2		27		102				walking difficulty	IVIG	improved
2	f	10yrs	30	139		5.5		9		28		104				breathlessness	nebulisation	improved
3	f	4.5yrs	15	137	127	3.72	3.2	7.5	8.2	11.5	11.2	103	99			vomiting f/b seizures	AED	improved
4	f	6yrs	20	139	134	5.5	4.9	10.2	10.1	26	24	98	98			fever with seizures	AED,antimalarial	improved
5	m	2.5yrs	12	131		4.1		9.2		22		102				cut injury lt elbow	antibiotics	improved
6	m	10yrs	24	132		4		10.1		26		102				vomiting f/b seizures	AED	improved
7	m	1yr	7	135	130	6.4	6.6	10.2		24	24	96	101			fever with seizures,vomiting	AED	improved
8	m	52 days	4	124	135	5	4	9.2		22		95				myocarditis,?AEFI	IVF,antibiotics	improved
9	m	7 months	7.5	135		5.2		10.1		23		107				septic shock,acute CNS inf	IVF,antibiotics,inotropes	improved
10	f	4.5yrs	15	137	144	3.72	5.3	10		11.5		103	99	resp alk with acidosis	Na-182,K-13.6,Cl-166	seizures/status epilepticus	AED,antimalarial	improved
11	m	45 days	2.5	134		10.2	6.2	9		28.2		98		normal		LOS,FTT,laryngomalacia	antibiotics	improved
12	m	9yrs	28	142		4.4		8.2		27		102				massive pleural effusion	ICD,ATT	DEATH
13	f	8yrs	30	144	138	3.8	4.2	9.1		23		103				?hypertensive encephalopathy	antibiotics,antimalarial,AHT	improved
14	m	5yrs	19	146	136	4.2	3.8	9.2		26		102				AGN with pulmonary edema	antibiotics	improved
15	f	10months	10	137		4.1		8.4		24		99				bronchopneumonia	IVF/antibiotics	improved
16	m	1.25yrs	9	141		5.2		8.1		14.7		99		metabolic acidosis		dilated cardiomyopathy	antifailure drugs	improved
17	m	8yrs	7.5	143		4		8.2		22		99				EHBA operated	IVF DNS	DEATH
18	m	6months	5.5	136		3.8		9		24		102				metabolic disorder	IVF DNS	improved
19	f	3yrs	15	141		4		8.4		16.5		99		metabolic acidosis		Paraquat poisoning	IVF	DEATH
20	m	4yrs	20	139	118	3.8	3.1	8.2		15.3		102		metabolic acidosiswith resp acidosis		Paraquat poisoning	IVF	DEATH
21	m	6.5yrs	20	139	138	3.3	4.3	8.2		26		101				snake bite	IVF DNS	improved
22	m	7yrs	21	137		4.2		9.1		27		99				GBS	IVF	improved

case no	gender	age	wt in kg	serum sodium		serum potassium		serum calcium		serum bicarbonate		serum chloride		ABG analysis	urine electrolyte	clinical history	hospital procedures			outcome
				initial	repeat	initial	repeat	initial	repeat	initial	repeat	initial	repeat							
23	f	9yrs	24	127	127	4.1		9.2		26		99				seizure/meningoencephalitis	IVF DNS/antibiotics			improved
24	m	2.5yrs	10	138	138	4.2	3.4	8		22		98		metabolic acidosis		multiple injuries head	antibiotics,AED			
25	f	11yrs	26	123	123	3.9	3.5	8.3		26		101				sickle cell anemia		antibiotics,antimalarial		improved
26	f	5months	6	134	134	4.9		9		24		98				FTT,choledochal cyst		antibiotics,vitamins		improved
27	m	2m0nths	3.9	135	135	5.7	4.5	8.2		26		102				bronchopneumonia,septic shock			IVF,inotropes,ventilated	DIED
28	m	8yrs	30	138	138	4.2		11		20.5		99		respiratory acidosis		drowning			inotropes, ventilated	improved
29	f	9yrs	24	138	138	4.1	3.5	10.2		22		106				seizures/NCL			AED,inotropes	improved
30	m	3yrs	9	124	124	5.4	4.6	8.1		24		101				SAM/TBM/Tbabdomen			IVF,ATT, antibiotics	improved
31	m	9months	7	138	138	3.8		8.2		26		107				bronchopneumonia,septic shock			inotropes, ventilated	
32	m	12yrs	26	139	139	4		9.1		28		102				seizures/NCL			inotropes, AED	
33	f	1yr	5	141		4.9		8.6				101				hydrocephalus/postvp shunt/septic shock	IVF,antibiotics			
34	f	52days	4.5	140		5.7		11.7				104				acute CNS infection	IVF DNS,antibiotics		inotropes,	improved
35	f	7yrs	15	133	141	3	4.2	9.2				101				acute CNS infection	IVF DNS,antibiotics			improved
36	f	4yrs	12	141		3		9				98				unknown poisoning	IVF,antibiotics			improved
37	f	5yrs	15	143	141	2.9	3.9	9.4				102				unknown poisoning,rat killer		IVF DNS,antibiotics		improved
38	f	10yrs	20	139		5.1		8.4				105				unknown poisoning		IVF DNS,antibiotics		improved
39	m	5yrs	15	141		3.4		9.2				104				unknown poisoning		IVF DNS,antibiotics		improved
40	m	1.4yrs	9	146		3.7		9				102				bronchopneumonia,septic shock		IVF,antibiotics		improved
41	f	7months	6	134		5.5		8.4				100				seizure disorder,Sturge weber syndrome		IVF,antibiotics		improved
42	m	4months	6	148	139	5.1	3.9	9				98				septic shock		IVF,inotropes,antibiotics		improved
43	m	4yrs	13	138		4.6						104				hepatoblastoma		IVF,antibiotics		improved
44	f	9yrs	12	133	141	5.4	4.8	9				9.8				DD,aspiration pneumonia		IVF,Inotropes,antibiotics		improved
45	f	7months	6.5	133		4.2		9.2				104				post traumatic seizures,		IVF DNS,antibiotics		improved
46	m	11yrs	28	130		3.4		9				100				acute CNS infection		IVF DNS,antibiotics		improved

case no	gender	age	wt in kg	serum sodium		serum potassium		serum calcium		serum bicarbonate		serum chloride		ABG analysis	urine electrolyte	clinical history	hospital procedures	outcome
				initial	repeat	initial	repeat	initial	repeat	initial	repeat	initial	repeat					
47	m	9yrs	23	134		4.5		9				101				acute dysentry, drug reaction	IVF,antibiotics	improved
48	f	3months	3.5	134	145	4.4	3.7	8.2				100	102			heart disease/ VSD/cardiogenic shock	IVF,inotropes,antibiotics	improved
49	m	4yrs	13	136		4		9.2				105				rt Hemothorax	IVF,inotropes,antibiotics	improved
50	f	1.2yrs	8	138	139	2.4	3.8	8				96	102			refractory seizures, shock	IVF ,inotropes,antibiotics	DEATH
51	m	3.5yrs	15	137	123	4.4	3	9.2	9	18.5		106	101		201	seizures/ febrile status epileptic	IVF,inotropes,antibiotics	improved
52	m	12yrs	26	139		4		8.9				107				seizure disorder, status epilepticus	IVF,Inotropes,antibiotics	improved
53	m	9months	7	138		3.8		9				102				bronchopneumonia,septic shock	IVF,inotropes,antibiotics	improved
54	m	7yrs	18	140		3.4		9				98				hepatic encephalopathy	IVF DNS,antibiotics	improved
55	m	9months	9	138		3.8		9.2				107				bronchopneumonia,septic shock	IVF,intubated,inotropes,antibiotics	improved
56	f	5yrs	24	136		4.2		9				102				Seizure disorder, status epilepticus	IVF,antibiotics	improved
57	m	2months	4.2	138		5.1		8.7				103				late onset sepsis,Seizure disorder	IVF,antibiotics	improved
58	f	10months	3	129		5		8.7				104				bronchopneumonia	IVF,antibiotics	improved
59	m	3yrs	18	144		3.2		8.1				102				DD/Seizuredisorder	IVF DNS,antibiotics	improved
60	m	5months	7	136		4.2		9.1				102				bronchopneumonia,septic shock	IVF,intubated,inotropes,antibiotics	improved
61	f	12yrs	30	136		4.2		8.8				111				septic shock	IVF,inotropes,antibiotics	improved
62	m	9months	9	138	136	3.8	4	9				107				bronchopneumonia,septic shock	IVF,inotropes,intubated	improved
63	m	1oyrs	26	141		4.2		8.8				102				Dengue,rt sided empyema	IVF DNS,antibiotics	improved
64	f	6yrs	10	139		3.9		8				104				spastic cp/aspiration pneumonia	IVF,inotropes,	improved
65	m	1.5yrs	10	135	145	3.8	3.4	9				111	103			fall from ht,seizures	IVF,NS,Inotropes	improved
66	f	43days	4.2	159	142	5.9	4.4	9.2				105				status epilepticus	IVF,NS,Inotropes	improved
67	f	31ays	3.2	131	139	5.8	4.7	9				101				late onset sepsis,Seizure disorder	IVF,antibiotics	improved
68	m	1.5yrs	10	137		4.5		8.8				102				bronchopneumonia,septic shock	IVF,inotropes,intubated	improved
69	m	5yrs	16	124	135	4.1	3.9	8		20.2		98	105			status epilepticus,SD	IVF,DNS,inotropes	improved
70	f	3yrs	12	145		4		9				98	102			poisoning/liquid detergent	IVF,inotropes	improved

case no	gender	age	wt in kg	serum sodium		serum potassium		serum calcium		serum bicarbonate		serum chloride		ABG analysis	urine electrolyte	clinical history		hospital procedures	management	outcome
				initial	repeat	initial	repeat	initial	repeat	initial	repeat	initial	repeat							
71	m	45days	4	137		5.8		9.2				102				bronchopneumonia /septic shock		IVF,intubated, inotropes		improved
72	f	5months	4	124	139	5	4.7	8.8				104				seizures disorder		IVF,antibiotics		improved
73	m	1.5yrs	10	138		5.2		9				102				poisoning liquid detergent		IVF,antibiotics		improved
74	f	3.5yrs	14	141	126	4.6	5.5	9.6				102				acute meningoencephalitis		IVF DNS/inotropes/intubated		improved
75	f	10months	3	129		5		8.7				104				bronchiolitis		IVF,intubated, inotropes		improved
76	m	9months	5.6	136		4.5		9.2				101				bronchopneumonia /VSD operated		IVF,antibiotics		improved
77	m	10yrs	30	130	133	5.4	4.9	8.8				102				viral hemorrhagic fever		IVF,inotropes, antibiotics		improved
78	m	1.5yrs	10	141	139	2.9	4.5	9				104				AGE/Septic shock/status epilepticus		IVF,intubated, inotropes		improved
79	m	34days	4.5	137	140	5.1	3.8	8.8				103	104			late onset sepsis/cardiogenic shock		IVF,inotropes, antibiotics		improved
80	f	12yrs	20	139		4.4		9				102				Turners syndrome /viralhemorrhagic fever				improved
81	m	2yrs	10	138		4.2		9				104				ADD /Viral fever		IVF,antibiotics		improved
82	m	11yrs	30	120	148	3.8	3.9	9.2				105				Addisons disease/Viral encephalitis		IVF,DNS		improved
83	f	4yrs	16	139		3.8		8.4				100				viral hemorrhagic fever		IVF,antibiotics		improved
84	f	2.5yrs	7	138		4.2		9.2				102				seizures disorder		IVF,antibiotics		improved
85	m	12yrs	30	141		3		9.2				102				seizures disorder		IVF,antibiotics		improved
86	f	8yrs	20	139		4.1		9				101				viral hemorrhagic fever		IVF,antibiotics		improved
87	m	1.5yrs	10	141		3.6		8.8				102				poisoning /kerosene ingestion		IVF		improved
88	m	2yrs	11	136		4		9.2				104				poisoning /kerosene ingestion		IVF		improved
89	f	9yrs	28	142		3.7		8.8				104				poisoning /acid ingestion		IVF,antibiotics		improved
90	m	2yrs	12	134		4.2		9				102				poisoning /camphor ingestion		IVF		improved
91	f	1.5yrs	10	139		3.6		10				102				bronchopneumonia /septic shock		IVF,antibiotics		improved
92	f	9yrs	29	141		3.8		9.2				101				scorpion sting		IVF		improved
93	m	2.75yrs	12	136		3.5		8.9				102				poisoning /acid ingestion		IVF,antibiotics		improved
94	m	9yrs	28	135		4		9.2				102				scorpion sting		IVF		improved

case no	gender	age	wt in kg	serum sodium		serum potassium		serum calcium		serum bicarbonate		serum chloride		ABG analysis	urine electrolyte	clinical history	hospital procedures	outcome
				initial	repeat	initial	repeat	initial	repeat	initial	repeat	initial	repeat					
95	f	4yrs	15	134	141	3.7	4.1	7.8				103				poisoning camphor ingestion	IVF,antiepileptics	improved
96	f	1.5yrs	12	136		3.8		10.1				102				poisoning acid ingestion	IVF,antibiotics	improved
97	f	5yrs	19	134	142	3.8		9				108				poisoning kerosene ingestion	IVF,antibiotics	improved
98	m	1.5yrs	11	139		4.1		10				102				poisoning camphor ingestion	IVF	improved
99	m	2yrs	12	141		3.7		9				102				poisoning tablet cyproheptidine	IVF	improved
100	m	1.5yrs	12	136		4.2		11				98				poisoning ratkiller	IVF	improved
101	f	1.5yrs	11	142		3.5		8.8				102				poisoning kerosene ingestion	IVF,antibiotics	improved
102	m	1yr	9	135		4.1		9				102				poisoning oil	IVF,antibiotics	improved
103	m	3yrs	12	132	141	3.6	3.9	8.2				102				poisoning phenyl	IVF,antibiotics	improved
104	f	2.75yrs	13	137		4.1		9				98				poisoning kerosene ingestion	IVF,antibiotics	improved
105	f	2.75yrs	12	136		3.6		10.2				101				poisoning diesel	IVF,antibiotics	improved
106	m	1.25yrs	9	141		3.2		9				98				bronchopneumonia/Septic shock	IVF,antibiotics	improved
107	m	11months	9	141		4.1		10.2				102				dilated cardiomyopathy	IVF,inotropes,antibiotics	improved
108	m	9yrs	30	136		3.6		9				105				Acute rheumatic fever with CCF	IVF,antifailure drugs	improved
109	m	2yrs	10	139		4.7		10.2				107				poisoning kerosene ingestion	IVF,antibiotics	improved
110	f	3yrs	11	136		4.2		9.2				104				poisoning tablet cyproheptidine	IVF	improved
111	m	2yrs	12	140		3.7		10				104				poisoning thinner ingestion		improved
112	m	11yrs	30	137		4.1		9				102				poisoning unkown tablet	IVF	improved
113	f	12yrs	27	136		3.4		8.9				102				poisoning ratkiller	IVF,antibiotics	improved
114	m	1.5yrs	9	136		4.1		10.2				102				poisoning ratkiller	IVF,antibiotics	improved
115	m	3yrs	14	133		3.7		10				99				poisoning antkiller	IVF,antibiotics	improved
116	m	4yrs	12	144		3.7		9				101				poisoning antkiller	IVF	improved

117	f	1.25yrs	9	135	136	5.1	4.8	10				99				poisoning kerosene ingestion	IVF,antibiotics	improved
118	m	1yr	10	141		3.6		8.8				100				poisoning kerosene ingestion	IVF,antibiotics/intubated	DEATH
119	f	4yrs	15	131		3.8		10.2				102				poisoning iron tablet	IVF,antibiotics/intubated	DEATH
120	m	8.5yrs	7.5	143		4		9.2				104				chronic liver disease	IVF,antibiotics,inotropes/intubated	DEATH
121	m	6months	5.5	136		3.8		10.1				99				metabolic disorder	IVF/antibiotics	improved
122	m	2months	3.9	135	138	5.7	4.6	9.2				102				bronchopneumonia/Septic shock	IVF/inotropes/intubated	DEATH
123	f	1.5yrs	9.7	148	158	4	3.4	9		13		100				GDD/MODS/IEM	IVF/inotropes/intubated	DEATH
124	m	7months	7	136	120	4.9	2.8	8				102				bronchopneumonia/supraventricular tachycardia	IVF/inotropes/intubated	DEATH
125	m	1yr	8	132	138	4.1	4.3	9				104				sialidoses/GDD/SD	IVF/inotropes/intubated	DEATH
126	f	12yrs	30	134	142	3.2	4.4	8				96				central/peripheral demyelinating disease	IVF	improved
127	f	11yrs	32	137		3.5		9.2				102				Seizure disorder/breakthrough seizure	IVF/antibiotics/antiepileptics	improved
128	m	7yrs	24	134	139	4.1	3.6	10				104				Guillain barre syndrome	Immunoglobins	improved
129	m	8yrs	22	138		3.7		8.8				100				RHD/CCF	antifailure drugs	improved
130	m	1.5yrs	9	139	141	3.4	4.4	8				102				poisoning/nitrazepam	IVF	improved
131	m	7yrs	24	137		4.1		9.2				101				viralhemorrhagicfever	IVF	improved
131	m	10yrs	30	136		3.5		10.1				98				viralhemorrhagicfever	IVF/inotropes	improved
132	m	12yrs	32	137		4.1		9.8				104				stevenjohnson syndrome	IVF	improved
133	m	1.5yrs	10	134	138	3.5	4	11.1	10			98				poisoning kerosene ingestion	IVF,antibiotics	improved
134	f	8yrs	26	137		4.3		8	9.2			96				poisoning tablet alprax	IVF	improved
135	f	11months	10	138		3.6		9				101				poisoning phenyl	IVF,antibiotics	improved
136	m	7yrs	29.5	134	142	3.8	3.2	8				98				hepatic encephalopathy	IVF/inotropes/intubated	DEATH
137	m	8yrs	28	136		4		9.2				102				drowning	IVF/inotropes/intubated	improved
138	f	2.5yrs	12	141		3.7		9				102				bronchopneumonia/septic shock	IVF/inotropes	improved
139	f	5yrs	8	132	131	2.8	3.4	8				104				GDD/FTT/SD	IVF/inotropes	DEATH
140	m	9months	8	141		3.6		10				98				septic shock	IVF/inotropes/intubated	improved
141	m	11months	10	137		4.4		9				99				hepatoma operated	IVF	improved
142	f	6yrs	20	141		3.8		9.2				98				seizure disorder/breakthrough seizure	IVF/antiepiletics	improved
143	m	7yrs	22	136	142	3.2	4.1	10				102				status asthmaticus	IVF/nebulisation	improved
144	f	3yrs	14	144		4.6		9.2				104				septic shock	IVF/inotropes	improved
145	m	7yrs	26	138		4.5		7.8	8.4			96				viralhemorrhagicfever	IVF/inotropes	improved

146	f	42days	3.5	139		3.6		8.8				102				late onset sepsis	IVF/antibiotics	improved
147	f	6yrs	18	134	142	3.8	4	9				104				GDD/seizure disorder	IVF/antiepiletics	improved
148	f	8yrs	28	135		4.2		10				102				sickle cell anemia	IVF/antibiotics	improved
149	m	9yrs	27	136		4.1		9				104				Diabetic ketoacidosis	IVF/inotropes/insulin	improved
150	f	6yrs	20	142		3.8		10				98				scorpion sting	IVF	improved
160	m	33days	3.5	134		4.1		9				102				bronchopneumonia,septic shock	IVF,inotropes/intubated	improved
161	f	1yr	10	136	141	3.2	4.1	10				98				sturgeweber syndrome	IVF/inotropes/intubated	improved
162	f	9yrs	31	134	137	4.1	3.6	8				102				spastic cp/aspiration pneumonia	IVF/inotropes/intubated	improved
163	f	8yrs	29	145		3.8		9				102				AGN/DCM	IVF/inotropes/intubated	improved
164	f	2yrs	12	135		4		8				99				bronchopneumonia/B/L Pneumothorax/septic shock	IVF/inotropes/intubated	DEATH
165	m	4months	7	136		3.5		9				102				bronchopneumonia/septic shock	IVF/inotropes/intubated	improved
166	m	5yrs	19	137	136	3.2	4.1	10				104				seizure disorder/status epilepticus	IVF/inotropes/intubated	DEATH
167	m	1.5yrs	8	133	135	4	3.8	8				98				DCM/refractory cardiogenic shock	IVF/inotropes/intubated	DEATH
168	m	63days	3.8	142		3.8		8				102				hydrocephalus/ETV done/decompensated shock	IVF DNS/inotropes/intubated	DEATH
169	f	9yrs	26	135		3.6		9				99				neuronal ceroidal lipofuschinosis/refractory seizures	IVF/inotropes/intubated	DEATH
170	f	1.25yrs	9	140		4		10				98				superrefractory status epilepticus/DD	IVF/inotropes/intubated	DEATH

171	m	11yrs	30	137		3.5		9				98				food poisoning/hypotensive shock	IVF/inotropes/intubated	DEATH
172	f	2months	5	141		3.6		10				101				Downs syndrome/refractory seizures	IVF/inotropes/intubated	DEATH
173	m	1.5yrs	10	136	135	3.2	3.6	9				99				head injury	IVF DNS/inotropes/intubated	improved
174	m	2yrs	11	131	138	4	3.7	8				102				Seizure/status epilepticus	IVF DNS/inotropes/intubated	improved
175	f	1yr	9	141		3.5		9				105				varnish poisoning	IVF/inotropes/intubated	improved
176	f	3yrs	12	139		4		8				102				lindane poisoning	IVF/inotropes/intubated	improved
177	m	1yr	9	134	136	3.5	4	9.2				105				ADD/refractory septic shock	IVF/inotropes/intubated	DEATH
178	f	4yrs	15	135			4	8.8				102				DKA	IVF/inotropes/intubated	improved
179	m	1.4yrs	7	136		3.6		9				104				disseminated TB/immunodeficiency/FTT/broncopneumonia	IVF/inotropes/intubated	DEATH
180	m	1.5yrs	9	131	140	4	3.7	8				102				Cyanotic HD/Tricuspid atresia	IVF/inotropes/intubated	DEATH
181	f	35days	2.8	134	135	3.3	3.6	8				99				anorectal atresia/cong anomalies	IVF	DEATH
182	m	45days	3	135		4		7	8			98				Severe pulm HTN/HUN	IVF/inotropes/intubated	DEATH
183	m	4months	6.5	138		3.6		9				102				hydrocephalus/VP shunt done	IVF/intubated	DEATH
184	f	1.5yrs	10	135		4		9				101				bronchopneumonia/Septic shock	IVF/inotropes/intubated	DEATH
185	m	9months	9	132	136	4		8.8				102				bronchopneumonia/VSD/dysmorphic facies	IVF/inotropes/intubated	DEATH
186	m	9yrs	26	135		3.5		9				98				GDD/status epilepticus/neurodegenerative disorder	IVF/intubated	DEATH
187	f	3months	6	141		3.6		10.1				104				FTT/ADD with severe dehydration/septic shock	IVF/inotropes/intubated	DEATH
188	m	5months	7	136		4		9.8				98				bronchopneumonia/hypernatremia/Aspiration pneumonia		DEATH
189	m	2.5yrs	14	128	136	3.8	3.6	10				102				multiple injuries/battered baby syndrome	IVF/inotropes/intubated	DEATH
190	m	45days	3.2	135	136	3.5	4	11				99				bronchopneumonia/Septic shock	IVF/inotropes/intubated	DEATH
191	m	4months	3.25	130	135	3.1	3.6	9				100				septic shock/renal failure	IVF/inotropes/intubated	DEATH
192	m	3months	3.1	135		3.6		10				102				acute CNS infection	IVF/DNS/intubated	DEATH
193	f	4yrs	15	132	136	4	3.5	9				98				DCM/refractory cardiogenic shock		DEATH
194	m	10yrs	30	136		3.7		10				98				GDD/SD/hydrocephalus/VP shunt	IVF/intubated	DEATH
195	m	1.25yrs	10	141		3.5		9				106				Seizure disorder/GDD/status epilepticus	IVF/inotropes/intubated	DEATH
196	m	2months	6	136		4.1		8				102				bronchopneumonia/meningitis	IVF DNS/inotropes/intubated	DEATH
197	m	3.5yrs	15	141		3.6		9				98				acute meningoencephalitis	IVF DNS/inotropes/intubated	DEATH
198	m	2.5months	5	136		4		8				102				bronchopneumonia/Septic shock	IVF/inotropes/intubated	DEATH
199	m	2months	5	132	136	3.4	3.8	7.8				101				ADD/refractory septic shock	IVF/inotropes/intubated	DEATH
200	f	2.5yrs	14	135		4		10				98				status epilepticus/IEM	IVF/inotropes/intubated	DEATH

case no	gender	age	wt in kg	serum sodium		serum potassium		serum calcium		serum bicarbonate		serum chloride		ABG analysis	urine electrolyte	clinical history	hospital procedures	outcome
				initial	repeat	initial	repeat	initial	repeat	initial	repeat	initial	repeat					
201	f	6months	7	136		3.6		8				101				Acyanotic Heart disease/FTT/ pneumonia	IVF,inotropes	DEATH
202	m	1yr	9	137		3.5		9				98				GDD/Rickets/ Bronchopneumonia	IVF/inotropes/intubated	DEATH
203	f	4months	7	131	138	3.7	4	9				102				hypothyroidism/Recurrent hypoglycemia/dysmorphic facies		DEATH
204	f	1yr	10	136		4		8				98				metabolic acidosis/IEM	IVF/inotropes/intubated	DEATH
205	m	10yrs	31	129	137	3.2	3.8	9				102				status epilepticus/decompensated shock	IVF/inotropes/intubated	DEATH
206	f	1.5yrs	10	136		4		8.9				98				kerosene ingestion	IVF	improved
208	m	10months	8.5	138	136	3.1	3.5	9				102				GDD/broncho pneumonia/decompensated shock	IVF/inotropes/intubated	DEATH
209	m	69days	5.2	135		4		10				98				septic cardiogenic shock	IVF/inotropes/intubated	DEATH
210	f	2yrs	12	136	135	3	4	9				99				CHD/infective endocarditis	IVF	DEATH
211	f	2.5yrs	11	139		4		8				102				GDD/broncho pneumonia/decompensated shock	IVF/inotropes/intubated	DEATH
212	m	1.5yrs	12	136		3.6		9				104				Headinjury	IVF DNS/inotropes/intubated	improved
213	f	3yrs	13	135		4		8				98				lindane poisoning	IVF/inotropes/intubated	improved
214	f	4yrs	13	136		3.6		9				102				varnish poisoning	IVF/inotropes/intubated	improved
215	f	11yrs	30	135		4		10				98				seizure disorder/status epilepticus	IVF/inotropes/intubated	improved
216	m	2.5yrs	12	132	136	3.7	3.5	9				102				phenyl poisoning	IVF	improved
217	f	5yrs	18	136	135	3.1	4	8				98				poisoning kerosene ingestion	IVF	improved
218	m	2.5yrs	12	135		3.5		9				100				poisoning liquid detergent	IVF	improved
219	m	4months	6	136		4		8.8				98				acute febrile illness/hypoxic posturing	IVF/inotropes/intubated	improved
220	f	9yrs	26	141		3.5		9				96				cerebral palsy/Aspiration pneumonia	IVF/inotropes/intubated	improved
221	m	8yrs	32	134	138	4		8				102				unknown poisoning	IVF DNS	improved
222	f	4yrs	22	138		3.9		9				100				turpentine poisoning	IVF	improved
223	m	2.5yrs	12	135		4		10				98				kerosene ingestion	IVF	improved
224	f	4months	5.5	136	137	3.1	3.6	9				102				seizure disorder/refractory seizures	IVF/inotropes/intubated	improved
225	m	1.5yrs	9	135		4		8				99				bronchopneumonia/Septic shock	IVF/inotropes/intubated	improved
226	m	2.5yrs	12	132	135	3.8	4	9				98				liquid detergent poisoning	IVF	improved
227	f	1.5yrs	10	141		4.2		8				102				bronchopneumonia/Septic shock	IVF/inotropes	improved
228	m	1.25yrs	9	132	136	3.5	4	9				98				seizures/breakthrough seizures	IVF/inotropes/intubated	improved
229	m	3yrs	13	140	142	3	3.5	8				102				head injury	IVF/inotropes/intubated	DEATH
230	m	45days	5.2	136		4		9				101				purpura fulminans/sepsis	IVF	improved
231	m	4yrs	7	135		3.5		9				102				hydrocephalus/ETV done/FTT	IVF	improved
231	f	2yrs	12	132	136	4	3.5	8				98				acute CNS infection	IVF DNS/antiepileptics	improved
232	m	1.5yrs	10	136		3.5		9				101				poisoning kerosene ingestion	IVF	improved
233	f	9months	9	138		3.5		9				102				intusussception operated	IVF	improved
234	m	2yrs	12	135		4		11.2	9.8			98				poisoning kerosene ingestion	IVF	improved
235	f	2.5yrs	13	136		3.5		9				102				bronchopneumonia/septic shock	IVF/inotropes	improved

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Clinical profile , Etiology, management and outcome of serum electrolyte disturbances in children admitted in PICU in tertiary care centre.

Principal Investigator : Dr. J Balaji.

Designation : PG MD (Paediatrics)

Department : Department of Paediatrics
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.01.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

K Vasantha

MEMBER SECRETARY,
IEC, SMC, CHENNAI

MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.